

A STUDY ON SWASAKASAM

Dissertation Submitted To

**THE TAMIL NADU DR.M.G.R Medical University
Chennai – 32**

For the Partial fulfillment in Awarding the Degree of

DOCTOR OF MEDICINE (SIDDHA)

(Branch – I Pothu Maruthuvam)



Department of Pothu Maruthuvam

Government Siddha Medical College

Palayamkottai – 627 002

SEPTEMBER 2008

ACKNOWLEDGEMENT

First, I thank **Almighty** for his showered blessings which is the ultimate source of my success.

I have to give thanks to our **Siddhars** for all their manifold mercies.

I wish to express gratitude and acknowledgement to the **Vice-Chancellor**, The TamilNadu Dr.M.G.R. Medical University, Chennai, The **Special Commissioner**, Director of Indian Medicine and Homeopathy, Chennai and **Joint Director** of Indian Medicine and Homeopathy, Chennai.

I also wish to convey my deep gratitude to **Prof. Dr. M.Dinakaran M.D.(s)**., Prinicipal, Government Siddha Medical College, Palayamkottai for patronizing the work by providing all the necessary facilities.

I also wish to convey my deep gratitude to **Prof. Dr.R.Devarajan M.D.(s)**., Vice Prinicipal, Government Siddha Medical College, Palayamkottai for patronizing the work by providing all the necessary facilities.

I express my deep sense of gratitude to **Prof.Dr.A.Prema M.D.(s)**., Head of the Department, P.G. Pothu Maruthuvam, Government Siddha Medical College, Palayamkottai for her valuable guidance and suggestion in caring out the dissertation.

I express my deep sense of gratitude to **Prof.Dr. K. R. Revathi M.D. (S)**, Former Vice Principal and Head of the Department P.G. Pothu Maruthuvam, Government Siddha Medical College, Palayamkottai, for her guidance and suggestions in the selection of topic.

I am extremely grateful to Reader **Dr.S.Mohan M.D.(s)**, and Assistant Lecturer **Dr.S.Chitra M.D.(s)**, Department of P.G Pothu Maruthuvam, Government Siddha Medical college, Palayamkottai for their kind and affectionate encouragement to my work.

I express my deep sense of gratitude to **Prof. Dr.S.Mohan M.D.**, Modern Medicine, Government Siddha Medical College, Palayamkottai for his Valuable guidance in modern aspects.

My special thanks goes to **Dr.D.Rajakumari M.D.(S)**, **Dr. P.Shanmugam M.D.(s)**, and **Dr.J.Angeline Nirmala M.D.(S)**, Assistant lectures, Department of U.G. Pothu Maruthuvam, Government Siddha Medical College, Palayamkottai for their timely help in referring cases for my dissertation work.

I render my special thanks to **Dr. S. Justus Antony M.D.(S)**, former Assistant lecturer, Department of P.G Pothu Maruthuvam, Government Siddha Medical College, Palayamkottai for his help in preparation of the drug.

I take this opportunity to express my deep sense of gratitude to **Lecturer. Mr. M.Kalaivanan M.Sc., M.Phil.**, and other staffs of Modern Pharmacology Department, Government Siddha Medical

College, Palayamkottai for their help during the entire course of my work.

I also thanks to **Prof. Mrs.N.Nagaprema M.Sc. M.Phil.,** Head of the Department and all the staffs of Department of Bio-Chemistry, for their help in Biochemical Analysis for this work.

On this occasion, I am thankful to **Dr.S.Bagirathi M.B.B.S.,M.D.,** Head and other staffs of Department of Clinical Pathology and **Dr.V.S.Padma M.B.B.S., D.M.R.D.,** along with technicians of Radiology Department for giving a kind Co-operation in doing investigation procedures.

I Cordially register my thanks to the Librarian **Mrs.T.Poonkodi M.A., M.Lisc., M.Phil.,** and Library staff, who helped to utilize the books of library for this work.

I express my thanks to **Dr.R.Napolean B.Sc., M.D.,** Consultant Microbiologist, Malar Micro Diagnostic Centre, Palayamkottai to evaluate anti-microbial activity of the trial medicine.

I would like to convey my thanks to **Aarthi Advanced CT Scan and MRI, Vannarpet, Tirunelveli,** for their Co-operation to diagnostic the disease.

I express my thanks cordially to **My Parents** for their Co-operation and Moral support from the very beginning of my career.

Finally I convey my thanks to Staffs, **Broad Band Net Café, (BBNC) Murugankurichi** for their expertise typing and printing work.

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INTRODUCTION

Every system of medicine promotes the adage “Prevention is better than cure”. Siddha system of medicine can help us to live a healthy life by preventing disease. This system is a preventive, protective and health promotive, curative, nutritive.

It is well known that all the eyes of the world are turning to the natural system of medicine. Especially indigenous system of medicine to find out a more acceptable drug for incurable disease.

Siddha system of medicine puts emphasis on three doshas. Any alteration in these three doshas will cause disease.

This can be understood from the verse as follows.

”மிகினும் குறையினும் நோய் செய்யும் நூலோர்
வளி முதலா எண்ணிய மூன்று”

- திருவள்ளுவர்.

Appropriate application of siddha science and system is sure to give us all a happy, healthy and harmonious life.

Maruthuvam (Medicine)

Medicine is one which prevents the disease of the body and mind and gives சநஅநனநைநள வழ னனைநயளந.

”மறுப்பது உடல்நோய் மருந் தெனலாகும்
மறுப்பது உளநோய் மருந்தென சாலும்
மறுப்பது இனிநோய் வாராதிருக்க
மறுப்பது சாவை மருந்தென லாமே”.

- திருமூலர்.

Through specific regulation of food consumption, one can prevent and cure the disease.

This is quoted in our system as follows.

“உணவே மருந்து மருந்தே உணவு”.

“மாறுபாடில்லாத உண்பி மருந்துண்ணில்
ஊறுபாடு இல்லை உயிர்க்கு”.

- திருக்குறள்.

The naturally available and scientific explanation of therapeutic efficacy of our drugs will convince many people to undertake our siddha system for their health needs.

SwasaKasam is a very common disease in the society due to increasing exposure to air pollution now a day. It is taken up for the Author's dissertation study. The increasing number of patients coming for treatment at Pothu maruthuvam out patient department and the absence of any permanent remedy in the allopathy system of medicine, has induced the author to select this disease for study.

The author has selected **Kasha Chooranam** (Anubava Vaithya Deva Ragaseum page-481) and **Anna pavala Chenduram** (Anuboga Vaitheya Navanethum part- III, page-104) as the trial drugs for the disease of swasakasam.

Kasha Chooranam, the ingredients of this medicine has a potent anti kasha action. Also the ingredients of Anna pavala Chenduram namely Anna Pethi and kodi pavalam has been recommended as a remedy for swasakasam in various siddha text. So, the author decided to undergo a trial on swasakasam with this powerful medicine.

AIM AND OBJECTIVES

Now-a-days people have vast knowledge about which system of medicine is better for the treatment of diseases depending upon their nature. They need a remedy which does not lead to complications and also they need a safe and permanent remedy, particularly for such chronic disease. The author has worked on SwasaKasam to get the patients treatment with out any side effects as this disease drives millions of people in difficulties to lead a normal day to day life.

To correlate the extensive study of SwasaKasam in siddha aspect as well as in modern aspect with bronchial asthma.

The daily increasing number of Asthma patients and the efficiency of siddha system of medicine curing chronic respiratory disease prompted the author to carry out scientific clinical study on the subject.

To make a detailed study of definition, etiology, classification, signs and symptoms, diagnosis, investigation, prognosis, line of treatment, diet control and prevention for SwasaKasam in various siddha literature.

To make a statistical study of the disease about its incidence with the criteria's like age, sex, socio-economic status, occupation, family history and paruvakaalam etc.

To study under the topics of mukkutram, poripulangal, udal thathukkal, envagai thervugal, naadi, neerkuri and neikkuri the changes brought about by this disease under normal conditions.

To make a detailed clinical evaluation of the disease on the basis of etiology, sign and symptoms, complications, treatment and progress during the course of disease.

To use modern parameters to confirm the diagnosis and to observe the prognosis of the patient.

To evaluate the Bio-chemical and pharmacological analysis and microbiological studies of the trial medicine.

To create an awareness among the public how the disease progresses on the basis of the factors like diet, land, climatic condition, pollution, immunology.

To educate the people who were affected by the disease how to stabilize their health through natural ways like pranayama, yogasanas, food restrictions and personal hygiene.

And also the prime object of this study is to do a clinical trial on SwasaKasam affected individuals with selected siddha Medicine.

Kasha Chooranam – 1gm and **Anna pavala Chenduram** – 100mg thrice daily with honey before meals.

ABSTRACT

Since the number of sufferers increasing day by day, the author has chosen the disease “SwasaKasam” for her dissertation work. The increasing incidence of the disease is due to changes in life styles and environment.

Twenty patients of either sex were selected as In-patients and twenty Out-patients were administered with the trial medicine “**Kasha Chooranam – 1 gm**” and **Anna pavala Chenduram – 100mg** with honey thrice daily before meals during whole study period.

The trial medicine was subjected to Bio-Chemical and Pharmacological as well as microbiological analysis.

At the end of the trial study, the majority of the cases showed good results.

REVIEW OF LITERATURE

SIDDHA ASPECTS

Veru Peyargal (Synonyms)

Swasa Irumal

Isivu Irumal

Izhuppu Irumal

Eyal (Definition)

Swasakasam is characterized by cough with expectoration, breath sound like hissing of snake, throat irritation, indigestion, flatulence, redness of the nose, low pitched voice, excessive salivation

Noi Varum Vazhi (Etiology)

Yugi Vaidhya Chinthamani Says,

“வேகின்ற வதிகமாம் புகையி னாலும்
மீறுகின்ற பாணத்தால் மிக்குந் தானே”

- 690

“பானத்தால் பரமாக்கினி மிசுக்கை யாலும்
பாரமா மாமிசங்கள் பசிக்கை யாலும்
தாணத்தாற் சஞ்சாரந் தவிர்க்கை யாலும்
சரிபடாப் பதார்த்தங்கள் பசித்த லாலும்
தீணத்தாற் பசியாம லிருக்கை யாலும்
சேயிழை யார்மேலின்பஞ் சிதைவ தாலும்
மானத்தாற் மாதுக்க மடைத லாலும்
மதத்தாலுஞ் சுவாசமது மருவுங் காணே”

- 691

Diet and habits

- ❖ Excessive smoking
- ❖ Excessive intake of cold water
- ❖ Increased body heat
- ❖ Excessive intake of non-vegetarian diet
- ❖ Lack of Exercise
- ❖ Taking improperly cooked food.
- ❖ Starving on hunger
- ❖ Mental stress

“காணவே தேவதைக்குப் பிரித்த பண்டம்
களவாடித் தின்றாலுங் கணவன்றன்னைத்
தோணவே நிந்தையைச் சொல்லுவதாலுஞ்
சுசியான பதார்த்த மெச்சில் பண்ணினாலும்
வேணவே ஒருவர் செய்த நன்றி தன்னை
மிக மறந்து கொடுமைகடான் விளம்புவோர்க்கும்
பேணவே சபைதனிலே சொன்னப் பேச்சுப்
புரண்டோர்க்குங் காசமது பிறக்குந் தானே”

Character and Behaviours

- ❖ Excessive Coitus
- ❖ Over Stress
- ❖ Stealing foods which were prepared for god
- ❖ Cursing life partner
- ❖ Tasting other's foods
- ❖ Forgetting one's help
- ❖ Those who didn't keep his words

Madhava Nidhanam @ Roga Vrichayam says,

புகையினால் சுவாச மார்க்கம் அடைபடுதல், ஆமரசம் சுவாசாசயத்தில் சேருதல், அதிக வியாயாமம் ருசியான அன்னத்தைப் புசித்தல், விழுங்கும் போது உணவு அதன் பாதையை விட்டு வேறு வழியில் பிரவேசிப்பது, வேகங்களை அடக்குவது, அவ்விதமே தும்மலை அடக்குவது.

- ❖ Excessive smoke
- ❖ Excessive gastric secretion and regurgitation
- ❖ Taking improperly cooked food
- ❖ Food enters into the larynx while swallowing
- ❖ Controlling reflexes like sneezing.

Roga Nirnaya Saram Says,

தேகத்தில் அக்கினி அதிகரித்து நெஞ்சு புண்ணாகி வாயு அதோமுகமாக மேல்சென்று மார்பில் தங்கி உண்டாகும்.

Due to excessive body heat, gas ascends to the lung field, thus causes the disease.

Siddha Maruthuvam (Pothu) Says,

இந்நோய் குளிர்காற்றிலீடுபடல், வெயிலில் மிகுதியும் அலைதல், மிக்க குளிர்ச்சியைத் தரும் பொருளையும், சூட்டைத் தரும் பொருளையும் உண்ணல், பெரிதும் உரத்துப் பேசுதல், மிக்க காரப்பொருள், நன்மணம், தீ மணம் ஆகியவற்றை முகர்வதாலும் பிறக்கும்.

- ❖ Exposure to cold weather
- ❖ Over strain in hot climate
- ❖ Taking cold and hot foods
- ❖ Singing in high pitched voice
- ❖ Due to irritants like dust, mud, lime etc.,
- ❖ Inhalation of pleasant as well as irritable odour.

Thanvandri Vaidhyam Says,

“அரசரோ கந்தனக்கே யமைச்சராய் காசரோகம்
தரை மிசை மாந்தர் தம்மைச் சார்ந்திடும் வகையோ தன்னி
லுரமிசை கிலேசந் தங்கு முறுதுய ராலு மாதர்
தருமயலாலுந் தூமஞ் சார்துகள் முகர்ந்ததாலும்.”

- ❖ Over stress
- ❖ Excessive Coitus
- ❖ Inhalation of dusts, pollens etc.,

Anubava Vaitheya Deva Ragaseyam says,

வாத கபங்களின் விருத்தி, அசீரண பேதி, வாந்தி, விஷப்பாண்டு, விடாச்சுரம், புகை, காற்று, தானியச்சுனை, அதிசீதளக்கபம், மர்ம தானங்களில் அடிபடுதல் முதலிய காரணங்களினால் உண்டாகும்.

- ❖ Excessive Vadha and Kaba.
- ❖ Diarrhoea due to indigestion.
- ❖ Toxic anaemia.
- ❖ Persistance fever.
- ❖ Excessive cold.
- ❖ Outer coat of the grains and pulse.
- ❖ Trauma in the genital organs.
- ❖ Pollution.

Murkurigal (Preliminary Signs)

தொண்டை புண்பட்டது போல நோதல், பின் தொண்டை சிவத்தல், தொண்டையில் முள்ளால் குத்துவது போன்ற உணர்ச்சி ஏற்படல், குரலோசை குறைதல், மூக்கு நீர் பாய்தல், மார்பு நோதல், சூடுள்ள பொருளில் விருப்பம் முதலியன உண்டாகும்.

- ❖ Sore throat
- ❖ Pricking sensation in the throat
- ❖ Decreasing the pitch of voice
- ❖ Running nose
- ❖ Pain in the chest
- ❖ Desire towards hot foods

Noi Enn (Classification)

Yugi Vaidhya Chindamani says,

Swasakasam is described as one of the twelve types of Kasam.

The twelve types are,

1. Mandhara kasam
2. Pakka Mandhara kasam
3. Sudar kasam
4. Vadha Kasam
5. Pitha Kasam
6. Swasa Kasam
7. Ratha Kasam
8. Silethuma Kasam
9. Peenisa Kasam
10. Vadhapitha kasam
11. Pitha silethuma kasam
12. Thontha kasam

Sikitcha Rathna Dheepam @ Vaidhya Chinthamani Says

There are twelve types of Kasam. They are

1. Mandhara Kasam
2. Patcha Mandhara Kasam
3. Sudar Kasam
4. Vadha Kasam
5. Pitha Kasam
6. Swasa Kasam
7. Ratha Kasam
8. Silethuma Kasam
9. Peenisa Kasam
10. Vadha pitha Kasam
11. Pitha Silethuma Kasam
12. Thontha Kasam

Raja Vaidhya Bodhini - Part I says

There are twelve types. Those are

1. Vadha Kasam
2. Pitha Kasam
3. Sethuma Kasam
4. Vadha Pitha Kasam
5. Pitha Sethuma Kasam
6. Mandhara Kasam
7. Swasa Kasam
8. Shaya Kasam
9. Sudar Kasam
10. Peenisa Kasam
11. Naadha Kasam
12. Thontha Kasam

Jeeva Rakshamirtham Says,

There are five types of Kasam. These are,

1. Vadha Kasam
2. Pitha Kasam
3. Silethma Kasam
4. Ratha Kasam
5. Shaya Kasam

Agasthiyar – 2000 Says

There are eight types. Those are

1. Vadha kasam
2. Pitha kasam
3. Kaba mandhara kasam
4. Pakka mandhara kasam
5. Mandhara kasam
6. Sudhika kasam
7. Marundheedu kasam
8. Kasam

Anubava Vaidhya Deva Ragasiyam Says,

There are five types. Those are,

1. Vadha Kasam
2. Pitha Kasam
3. Silethuma Kasam
4. Ratha Kasam
5. Shaya Kasam.

Tamilaga Siddha Vaitheya Gurugulam says,

There are twelve types. Those are.

1. Vadha kasam
2. Pitha kasam
3. Kaba kasam
4. Vadha pitha kasam
5. Pitha kaba kasam
6. Thontha kasam
7. Mandhara kasam
8. Patcha mandhara kasam
9. Ratha kasam
10. Peenisa kasam
11. Sudar kasam
12. Swasa kasam.

SWASA KASAM

Kuri Gunangal

The signs and symptoms are described in many siddha literatures. They are described as follows,

Yugi Vaidhya Chintamani Says,

“வண்மையாய் கோழை கட்டி இருமீ வீழும்
மாநாகம் போலவேவாங் குஞ்சு வாசம்
திண்மையாய் செருமலுண்டா மடிக்க டிக்குச்
சீரணயில் லாமலேவ யிறு ஊதும்
நண்மையாய் நாசியது தனல்போ லாகும்
நளிந்து டம்புவற்றிவரும் குரலுங் கம்மும்
உண்மையா யுண்ணாக்கி லூறுங் கேணி
உழுறுமே சுவாசகா சத்தி னொப்பே”.

According to Yugi Vaidhya Chinthamani the characteristic features of Swasakasam are cough with expectoration, breath sound like hissing of snake, throat irritation, indigestion, flatulence, redness of the nose, low pitched voice, excessive salivation.

Uyir Kakkum Siddha Maruthuvam @ Athma Ratchamirtham says,

உடல் உலர்ந்து வரும், சுரம், குளிர், இருமல், ஆயாசம், தலைவலி காணும். வயிறு பொருமி வாந்தி பண்ணும், மலம் கட்டி வியர்வை, தாகம் மிகும், புறந்தாள் அதைக்கும்.

Dryness of the skin, fever, rigor, cough, malaise, head ache, vomiting because of indigestion, sweating due to constipation, excessive thirst, pedal oedema present in Swasakasam.

Raja Vaidhya Bodhini - Part I says

வாத நாடியும், பித்த நாடியும் ஒருங்கு சேர்ந்து ஆமையைப் போல் மெல்ல ஊறி நடக்கில், வயிறு இரைச்சல், அன்னஞ் செரியாமை, கோழை கட்டல், இருமல், நாசி வரட்டல், குரற் கம்மல், முதுகெலும்பு எரிச்சல், மேற்சுவாசம்.

While the vadha pulsation and pitha pulsation is felt like the movement of tortoise, Swasakasam characters are flatulence, indigestion, mucoid sputum, cough, dryness of nose, low pitched voice, burning sensation along the vertebral column, dyspnea.

Tamilaga Siddha Vaitheya Gurugulam says,

அதிகமான கோழையுடன் இருமல், நாகப்பாம்பின் சீறலையொத்த சப்தத்துடன் சுவாசத்தில் சீற்றம், சீரணமின்மை, வயிற்றுப்பிசம், மூக்கில் அனலோடு வறட்சி, உடல் வற்றல், குரற்கம்மல், மூச்சு திணறல், உள்நாக்கில் வழுவழப்பான நீருறல், அளவுக்கு மீறிய இழுப்பு, இசிவு.

Cough with expectoration of large quantity of sputum, breath sound like hissing of snake, indigestion, flatulence, dryness and heat in the nose, emaciation, low pitched voice, viscous salivation, wheezing.

Mukutra Verupadugal (Pathology)

In siddha system of medicine, the manifestation of all the diseases are the result of derangement of Doshas i e., Vadham, Pitham, Kabam. The prime factor which is involved in Swasakasam is Kaba, which is accompanied with vitiated Vadha or Pitha so and produce clinical symptoms of Swasakasam. This is clearly indicated by Theraiyar as ,

“கபத்தினை யன்றி காச சுவாசம் காணாது”

- தேரையர்

1. Excess of Kaba in the respiratory organs affects the Melnokku kal and Uyir kal and so the vayu is not able to reach the terminal points of respiration which producing gasping and laboured breathing.
2. Some authors says that the disease is caused by deranged Vadha. This thought is also acceptable because the destruction of Vayu in the respiratory tract is abnormally present.
3. Excessive intake of Pitha prompting diet induces Pitha Kutram. This type of Pitha produce more heat and this heat goes to head resulting in running nose, heaviness of head and neck, sneezing and also induces formation of water vapours in the lungs and causing narrowing of air passage, which leads to the onset of the disease. This is indicated as,

“பித்தமே மிகுந்தா லீளை

யிருமலும் பெலத்து நிற்கும்”

- நோய் நாடல் நோய் முதல் நாடல்

So the changes in the diet and habits which increase Vadha and Kaba produces the clinical symptoms of Swasa Kasam.

In Uyir Nilaigal, Anagatham (chest) which is the residence of Udhanan (Melnokku kal) and Pranan (Uyir kal) is deranged.

When Pranan, the primary vayu is affected it leads to difficulty in breathing and involvement of Udhanan leads to cough and sneezing.

Involvement of Kirugaran leads to running nose, cough, sneezing. Involvement of Devathathan leads to tiredness. Involvement of Samanan causes inability to control the other Vayus and causes loss of appetite. Involvement of Sadhaga pitham leads to sluggishness. In Kaba, the derangement of Avalambagam leads to dyspnea, cough, wheezing. In the seven Udal Thathus, Saaram, Senneer are affected which leads to lethargy and depression. In severe cases Oon and Kozhuppu are also affected leads to symptoms of emaciation and body pain.

Piniyari Muraimai (Diagnosis)

The way of diagnosis is very important by which a physician can deal the disease, then only he will rule out the cause of the disease which is the main thing to be treated. Thiruvalluvar said,

“நோய்நாடி நோய்முத னாடியது தணிக்கும்
வாய்நாடி வாய்ப்பச் செயல்”

- திருக்குறள்

The diagnosis is based on four criterias

1. Poriyal arithal
2. Pulanal arithal
3. Vinathal
4. En Vagai Thervugal

1. Poriyal Arithal

Porigal are the five organs of perception. They are Eyes, Ears, Nose, Tongue, Skin. Poriyal Arithal is examining the Pori of the patient by the Pori of the physician. In swasaKasam, it is as follows,

Mei (Skin)	:	Normal
Vai (Tongue)	:	Excessive salivation
Kann (Eye)	:	Some times affected (redness)
Mookku (Nose)	:	Running nose
Sevi (Ear)	:	Normal

2. Pulanal Arithal

Pulungal are the five objects of senses.

Ooru (Sensation)	:	Warmth
Oosai (Sound)	:	Normal
Ozhi (Vision)	:	Normal
Suvai (Taste)	:	Normal
Naatram (Smell)	:	Altered or absent due to running nose and inflammation of nasal mucosa.

3. Vinadhal

By Vinadhal, the physician knows about the patient's Name, Age, Occupation, Native place(Thinai), Family history, Socio - economic status, Diet habits, Prone to any allergens, (ex: dust, smoke, pollens) His complaints, History of previous episodes, Frequency of attacks by changes in season, aggravates factors relevant history of treatment and Habits etc.,

Kaalam (Age Distribution)

The period of human life is totally 100 years. This is divided into three stages, according to the domination of three humours. As per this

1. Vadha Kaalam - 1 to 33 years
2. Pitha Kaalam - 34 to 66 years
3. Kaba Kaalam - 67 to 100 years

Even though in each of these stages, the other humours are also involved a particular humour is dominating more. According to this, Kaba types of diseases are more prone in the later stage (Kaba Kaalam).

Ivagai Nilangal

Study of Ivagai Nilangal is very important and useful because there may be possibility of the disease in some area. Ivagai Nilangal are,

- | | | |
|-------------|---|--------------------------------|
| A. Kurinchi | - | Mountains and its surroundings |
| B. Mullai | - | Forests and its surroundings |
| C. Marutham | - | plains and its surroundings |
| D. Neithal | - | Seas and its surroundings |
| E. Palai | - | Deserts and its surroundings |

Kurinchi

“குறிஞ்சி வரு நிலத்திற் கொற்றமுண்டி ரத்தம்
உறிஞ்சி வரு சுரமு முண்டாம்- அறிஞரைக்
கையமே தங்குதரத் தாமைவல்லை யுங்கதிக்கும்
ஐயமே தங்கு மறி.”

- பதார்த்த குண சிந்தாமணி

In Kurinchi Nilam, people are affected by fever that reduces blood level in the body, diseases related to spleen and liver and mainly by Kaba diseases.

Mullai:

“முல்லை நிலத்த மைய முந்நிரை மேவினுமவ்
வெல்லை நிலைத்த பித்த மெய்துறுங்காண்- அல்லவெனின்
வாதமொழி யாததனுண் மன்னு மவைவழி நோய்ப்
பேதமொழி யாதறையப் பின்பு.”

- பதார்த்த குண சிந்தாமணி

Though Mullai Nilam is the place of cattles, it is the place of increasing Pitha, Vadha also joined to that Pitha due to these Kutrams many diseases occur. It is difficult to distinguish between them.

Marutham

“மருதநில நன்னீர் வளமொன்றைக் கொண்டே
பொருதநில மாதியநோய் போக்குங் - கருதநிலத்
தாறிரதஞ் சூழ வருந்துவரென் றாற்பிணியெல்
ஏறிரதஞ் சூழ்விக்கு மில்”

- பதார்த்த குண சிந்தாமணி

Marutha Nilam, due to its water sources, cures all the three Vatha, Pitha and Kaba diseases.

Neithal

“நெய்தனில மேலுவர்ப்பை நீங்கா துறினுமது
வெய்தனில மேதங்கு வீடாகும் - நொய்தீன்
மருங்குடலை முக்காக்கி வல்லுறுப்பை வீக்குங்
கருங்குடலைக் கீழிறக்குங் காண்”

- பதார்த்த குண சிந்தாமணி

Through Neithal Nilam has the dominant taste of Uvarppu (Salty), it is the place of Pitha Vayu. The people who dwell here are susceptible to odema due to Kaba, Silipatha Rogam (Filariasis), Kudalanda Viruthi (Hernia).

Palai

‘பாலை நிலம்போற் படரைப் பிறப்பிக்க
மேலைநில மீயாது விரித்தற்கு - வேலைநில
முப்பிணிக்கு மில்லாம் முறையே யவற்றாலாம்
எப்பிணிக்கு மில்லா மஃதெண்”

- பதார்த்த குண சிந்தாமணி

The Palai Nilam is the place for grief and place of deadly Vadha, Pitha and Kaba diseases.

PARUVA KAALAM (Season)

As the earth revolves around the sun it gets sunlight at various positions. With reference to the position of the earth towards the sun, year is divided into six seasons.

They are,

1. Karkaalam (Avani & Purattasi) : August & September
2. Koothirkaalam (Iyppasi & Karthigai) : October & November
3. Munpanikaalam (Margazhi & Thai) : December & January
4. Pinpanikaalam (Masi & Panguni) : February & March
5. Elavenilkaalam (Chithirai & Vaigasi) : April & May
6. Mudhuvenilkaalam (Aani & Aadi) : June & July

According to literature, Swasakasam comes during rainy season (Karkaalam). In Koothirkaalam also due to cold wind there is increased incidence of disease.

Swasakasam mainly occurs due to vitiation of Kaba. Kabam thannilai sirappurum Kaalam - Karthigai to masi.

“மூவரு மீறி முனிவு கொளாமல்
தத்தம் நிலையில் தன்னரசியலும்
காலவரைதனை கிளரக் கேண்மின்
ஆடியாதியாய் ஐப்பசி ஈராய்
ஆனிலமதற்கோ ராசியல் காலம்
மீன் முதலாளி வீறுகொள் மந்திரி
தேள் முதன் மாசி சேனாபதிக்கே”

தேள் - கார்த்திகை

- நோய் நாடல் நோய் முதனாடல்

Hence the disease can occur in the later part of Koothir Kaalam to early part of Pinpani Kaalam, (i.e,) from the last two weeks of October to the first two weeks of February.

Totally the prevalence of disease is from August to February.

7. MUKKUTRA NILAIGAL

VADHAM

Pranan

It is responsible for respiration.

In Swasakasam, Vayu is affected leading to difficulty in breathing.

Abanan

It helps in excretion of urine and motion.

In Swasakasam, some patients had constipation.

Viyanan

It's main function is distribution of saaram.

Samanan

Samanan is the vayu that controls other vayus and digestion.

In Swasakasam, this vayu is affected since it cannot control the other vayus.

Udhanan

Its main function is inspiration and expiration and distributes the saaram equally to all tissues.

In Swasakasam, this vayu is affected due to difficulty in breathing.

Nagan

This vayu maintains opening and closure of eye lids and is not affected in Swasakasam.

Koorman

This vayu is responsible for vision and yawning.

Kirugaran

It causes salivation, running nose, sneeze, and cough and maintains appetite.

In Swasakasam, this vayu is deranged causing running nose, sneeze, cough and loss of appetite.

Devathathan

It is responsible for tiredness, anger and emotional expression.

In Swasakasam, this vayu is deranged causing emotional stress and insomnia.

Dhananjeyan

It produces swelling of the body after death and escapes through the scalp after the third day of death.

PITHAM

Anal pitham

This lives in the stomach and helps in digestion.

In Swasakasam, most of the patients complained loss of appetite and indigestion.

Ranjagam

This is residing in stomach and gives colour to the blood.

Sadhagam

It resides in the heart and executes the day to day activities with the help of mind and brain.

In this disease, restlessness, breathlessness present.

Aalosagam

It resides in both eyes and is responsible for clear vision.

Prasagam

It resides in skin and gives complexion.

In Swasakasam, some patients may have eczema with blackish discolouration of the skin.

KABAM

Avalambagam

It is residing in lungs and helps other four types of Kaba to function and also helps in the function of heart.

It is deranged in Swasakasam patients, since the presence of tightness of chest, cough, wheezing, and dyspnea.

Kilethagam

It is present in the stomach and gives moistures to the food materials and also helps in digestion.

In this disease, some patients have indigestion.

Pothagam

Living in the tongue and responsible for taste sensation, is not affected in Swasakasam patients.

Tharpagam

Living in the head and keep the eyes cooling.

In Swasakasam, there may be redness of eyes.

Sandhigam

It resides in the joint and helps for free movements.

UDAL KATTUGAL

Saaram

It is the energy part of end product of digestion.

It strengthens the body and mind. It is deranged in Swasakasam due to loss of appetite causing tiredness in the body and mind.

Senneer

It is responsible for knowledge, strength, boldness and healthy complexion. This is deranged in some patients with weakness.

Oon

It gives the structure to the body and is responsible for the movement of the body and is not affected in Swasa kasam.

Enbu

It gives the shape to the body and is responsible for motion of the body is not affected in Swasa kasam.

Kozhuppu

When the organs are doing their work this gives lubrication and facilitates their work and is not affected in Swasa kasam.

Moolai

It is present in the core of the bone which strengthens and maintains the normal condition of the bone, is not affected in Swasakasam.

Sukkilam / Suronitham

It is responsible for reproduction.

When the seven Udal Katukal increase or decrease from the normal level, the normal functioning of the body is affected.

4. EN VAGAI THERVUGAL

It is the basic diagnostic principle and the uniqueness of the Siddha system of Medicine. The following lines reveal this as follows.

“நாடிஸ்பரிசம் நாநிறம் மொழிவிழி

மலம் மூத்திரமிவை மருத்துவ ராயுதம்.”

- நோய் நாடல் நோய் முதனாடல்

And,

‘மெய்க்குறி நிறந்தொனி விழி நாவிருமலம் கைக்குறி”

- நோய் நாடல் நோய் முதனாடல்

The diagnostic value of EN VAGAI THERVUGAL is specific to Siddha system of Medicine and presumes the vitiated doshas in the patients.

En Vagai Thervugal are,

- a. Naa (Tongue)
- b. Niram (Colour of the skin)
- c. Mozhi (Speech)
- d. Vizhi (Eye)
- e. Malam (Motion)
- f. Moothiram (Urine)
- g. Sparisam (Palpation)
- h. Naadi (Pulse)

a. Naa

It is noted for its colour, ulcer, growth, coating, colour and consistency of the sputum that is spitted from mouth.

In Swasakasam, patients have the sputum scanty and mucoid.

b. Niram

Colour of skin, conjunctiva, and teeth.

In Swasakasam, the colour of the skin, conjunctiva, may be pale. In some patients, the conjunctiva may be red due to conjunctivitis.

c. Mozhi

Generally, speech is generated from the voice box. Abnormalities are low pitched speech, lalling, diplegia, monotonous speech, jerky, scanning, hot potato, indistinct, lisping.

In Swasa kasam mode of speech may be emotional or difficulty in speech, low pitched voice, wheezing sound is heard.

d. Vizhi

Type of eye - redness, ulcer, pallor, protrusion, tears, shedding of eye lashes, excreta of eye, diseases of eyes are noted.

In Swasa kasam, the eyes may be red.

e. Malam

Consistency hard or semisolid or diarrhoea, undigested food, fluid resembling the water used to clean meat, colour, frothy, dysentery, bloody, pus, mucous, smell, frequency of defaecation, constipation, reduced or increased stool content, lower abdominal pain during defaecation are noted.

In Swasa kasam, the patients may be constipated.

f. Neer @ Moothiram

Colour - yellow, black, white copper coloured, mixed colour, colour of fumes, smell-smell of fire, honey, sweet odours, fragrance of flowers, fruity odour, odour of deer flesh, frothy or not, frequency and quantity are noted.

In Swasa kasam it may be transparent and frothy.

g. Sparisam

Heat or coldness of the body.

It may be cold due to sweating in this disease.

h. Naadi

Naadi is the very important helpful observation for diagnosis and prognosis.

In **Noi Naadal Noi Mudhal Naadal** Text, Naadi is defined as,

“உடலில் உயிர் தரித்திருப்பதற்குக் காரணமான சீவசக்தி
எதுவோ அதுவே தாது அல்லது நாடி எனப்படும்”

Genesis of Naadi

The three Uyir Thathukkal are formed by the combination of three Naadis and three Vayus .

Idakalai + Abanan = Vatha

Pinkalai + Pranan = Pitha

Suzhumunai + Samanan = Kaba

This can be felt one inch below the wrist on the radial side by means of palpation by the three fingers - index, middle and ring fingers corresponding to vadha, pitha and kaba respectively.

“கரிமுகனடியை வாழ்த்திக் கைதனில் நாடிபார்க்கில்
பெருவிரலங்குலத்தில் பிடித்தடி நடுவே தொட்டால்
ஒரு விரலோடில் வாதமுயர் நடுவிரலிற் பித்தம்
திருவிரல் மூன்றிலோடில் சேத்தும நாடிதானே”

- அகத்தியர் நாடி.

Naadi Nadai in Swasa Kasam

Vatha Kaba Naadi

“பாங்கான வாதத்தில் சேத்தும நாடிப்
பரிசித்தால் திமிர் மேவு முளைச்சலாகும்
தீங்கான இருமலுடன் சந்தி தோடம்
சேர்ந்த விடம் வெடிசூலை யிருத்தோகம்
வாங்காத ஈளை மந்தார காசம்
வலியுடனே புறவீச்சுயள் வீச்சு வீக்கம்
ஓங்காணுஞ்சுர முடனே சுவாச காசம்
உண்டாகும் வெகு நோய்க்கு முறுதிதானே”

- சதக நாடி

Iya Naadi

“தானமுள்ள சேத்து மந்தானிளகில் வெப்பு,
சயமீளை யிருமல் மந்தார காசம்
ஈனமுறுஞ் சந்நிவிட தோடம் விக்கல்
யிருத்ரோகங் கரப்பான் விரண தோடம்
மானனையீர் குலைதிரள் வியாதி வீக்கம்
வருஞ்சக்தி சுவாசம் நெஞ்சடைப்பு தூக்கம்
ஏனமுறுங் காமாலை பாண்டு சோபை
ஏழுசுரங்கள் பலதுக்கம் விட முண்டாமே”

- சதக நாடி

Kaba Pitha Naadi

“இடமான சேத்துமத்தில் பித்த நாடி
எழுந்தணுகில் விடமுடனே வீக்கமுண்டாம்
திடமான குளிர் காய்ச்சல் மஞ்சள் நோவுந்
தேகத்தி லுளைச்சலிளைப் பிருமல் வாந்தி
விடமான நெஞ்சடைப்பு சுவாசம் விக்கல்
வெகு சுரமும் நாவறட்சி பாண்டுரோகம்
அடமான குவளைரத்த மதிசாரந்தான்
அணுகி வெகுபல நோய்க்குந் தடங்கண்டாயே”

- சதகநாடி

Iya Ushnam

“கதிப்பான சேத்துமத்திலுட்டிணங் கூடில்
கலந்த குணஞ்சயமிருமல் சுவாசகாசம்
மதிப்பான கோழைரத்தம் விப்புருதியுடனே
வளர்நாசிகா பீடமிருத் ரோகம்
கொதிப்பான சிங்ஙவையாக் கிராணவாயு
கொட்டாவி விக்கல் மந்தாரகாசம்
துதிப்பான வீரலத்திக் காய்வுரத்தம்
தோன்றுமிகு பிணிபலவுந் தொந்திப்பாமே”

- சதக நாடி

Iya Vayu

“தொந்தித்த சேத்துமத்தில் வாயுகூடித் தொடர்ந்த
குன்மம் நெஞ்சடைப்பு சுவாசகாகம்
வந்தித்த குரல்தனிலே உறுத்த லீளை
வழுவழுப்பு நீருறல் மலத்தில் சீதம்
வெந்தித்தல் கொழுத்தல் குத்துந் திமிர்வியாதி
வீச்சடனே வலியெட்டுந் திரட்சி பாண்ட
அந்தித்த கிறுகிறுப்பு மயக்கம் விக்கல்
ஆனபல பிணிகளுமே வந்தட ருந்தானே”

- சதக நாடி

Hence the Naadi Nadai in Swasa kasam is Kaba, Vadha kaba, Kaba Pitha, Iya Ushna, and Iya Vayu Naadis.

Nei Kuri

This urine examination is unique in Siddha system of Medicine. For this examination urine is collected in the early morning in a pure glass vessel. Patient is advised to take a balanced diet and avoid excessive diet, and in take of diet during irregular timings on the previous day of examination.

“அருந்துமாறி ரதமும் அவிரோதமதாய்
அகல் அலர்தல் அகாலவூன் தவிர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காது பெய்
தொருமுகூர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே”

- சித்த மருந்துவாங்கச் சுருக்கம்

“நிறக்குறிக் குரைத்த நிருமாண நீரிற்
சிறக்க வெண்ணெய்யோர் சிறுதுளி நடுவிடுத்
தென்றுறத் திறந்தொலி யேகாதமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுஞ் செய்தியை யுணரே”

- நோய் நாடல் நோய் முதனாடல்

A drop of gingelly oil is dropped on a wide glass vessel containing the urine to be tested which is kept under sunlight in a calm place. The derangement of the three dhoshas can be diagnosed by the mode of spread of gingelly oil on the surface of urine.

“அரவென நீண்டின் அ/தே வாதம்”

“ஆழிபோற் பரவின் அ/தே பித்தம்”

“முத்தொத்து நிற்கின் மொழிவதென் கபமே”

- நோய் நாடல் நோய் முதனாடல்

- ❖ Oil spreading like a snake indicates Vatha.
- ❖ Oil spreading like a ring indicates Pitha
- ❖ Oil spreading like a pearl indicates Kaba

“அரவிலாழியும் ஆழியில் அரவும்

அரவில் முத்தும் ஆழியில் முத்தும்

தோற்றில் தொந்த தோடங்களாமே”

- நோய் நாடல் நோய் முதனாடல்

Oil spreading like snake and ring, ring and snake, snake and pearl, ring and pearl all comes under Dhondha Dhosham.

In Swasa kasam, most of the Nei Kuri findings result- pearl like oil floating on the urine.

DIFFERENTIAL DIAGNOSIS

DISEASES SIMILAR TO SWASAKASAM are,

Mandara Kasam

“தானான தூயதோர் நாசி தன்னில்
சலநோய் நீர்தான் விழுந்த தும்மலுண்டாம்
மானான மார்புநெஞ் சடைத்து மூச்சு
வலுவாக பாம்புபோல் சீற லாகும்
கானான கண்டமோடு முகமுங் காதும்
காயமதுங் கசிவாகி வியர்வை யாகும்
ஏனான இருமலோடு கோழை கம்மல்
இரைப் பாகு மந்தாரகாச மாமே”

- யூகி வைத்திய சிந்தாமணி

In Mandara kasam there is running nose, sneezing, tightness of chest, breath sound like hissing of snake, sweating all over the body, cough, expectoration, dyspnea, etc.,

In Swasa kasam there is no sweating all over the body.

Kandakiragam

“வகையான குரலதனைப் பற்றி நொந்து
மார்போடு பிடரியினில் வலி யுண்டாகி
நுகரான சரீமெல்லாம் நொந்த ழாற்றி
நுணுக்கமாய்ச் சுவாசமது புறப் படாமல்
முகையான நாவாவே மூச்சு மாறி
முகத்திலே வியர்வாகி விலாநோ வுண்டாம்
புகையான வன்னத்தைப் பருகொட் டாது
பரியகண்ட கிரகத்தின் பண்பு தானே”

-யூகி வைத்திய சிந்தாமணி

In Kandakiragam, there is difficulty in speech, pain in the chest and occipital region, pain all over the body, breathlessness, buccal respiration, sweating in face, pain in the ribs, anorexia.

In Swasa kasam, there is no pain in the occipital region.

Swasa Pitham

“கருத்தாகச் சுவாசமது மிகவுண் டாகுங்
கனமாக வயிறுமே ஊதிக் காணும்
உருத்தாக உடலதுதான் மிகவ லிக்கு
மூறுமே கேணி போல் வாய்நீர் தானும்
மருத்தாக மயங்கியே கண்ம றைக்கும்
மார்பிலே வலியோடு இரும லுண்டாந்
துருத்தாக வயிறதனின் பசியோ வில்லை
சுவாசமாம் பித்தத்தின் சூட்சந் தானே”

- யூகி வைத்திய சிந்தாமணி

In Swasa Pitham, there is increased respiration (tachypnoea), flatulence, pain all over the body, brushing, loss of consciousness, pain in the chest followed by cough, loss of appetite etc.,

In Swasa kasam there is no loss of consciousness.

Swasa Silethumam

‘திறமையாய் நெஞ்சுதனிற் கோழை கட்டுஞ்
சிக்கென்று தானிருமி மூக்க டைக்குங்
குறுமையாய் குறட்டென்று சுவாசங் காணுங்
குளிரோடு சுரமுண்டாய் மயக்க மாகும்
மறமையாய் மார்போடு நெஞ்ச டைக்கும்
வாய்வறண்டு மூக்கதனில் நீரே பாயும்
வெறுமையாய் மிகத்தண்ணீர் தாப முண்டாய்
விடுசுவாச சிலேட்டுமத்தின் விவரந் தானே”

- யூகி வைத்திய சிந்தாமணி

In Swasa Silethumam, there is congestion in lungs, nasal congestion, cough, dyspnea, fever with rigor, syncope, tightness of chest, dryness of mouth, running nose, excessive thirst etc.,

In Swasa kasam, there is no fever with rigor, excessive thirst etc.,

Silethuma Vadha Suronitham

“பண்பாக வுடல்குளிர்ந்து வயிறு வீங்கிப்
பதைப்பான விடந்தொட்டாற் போல் நோவாந்
திண்பான சிரசு நெற்றி நோக்கா டுண்டாஞ்
சிலேட்டுமமாய்க் கோழையோடு சுவாசமாகும்
மண்பாக மயக்கமொடு கனவு முண்டாம்
வாய்வறண்ட ருசியில்லா வருத்த மாகும்
நண்பாக நாடியுமே படப் படக்கும்
நற்சிலேட்டம் சுரோணிதமாம் நாடுங் காலே”

- யூகி வைத்திய சிந்தாமணி

In Silethuma Vadha Suronitham, there is chillness of body, abdominal distension tenderness in abdomen, headache, expectoration, dyspnea, fainting, dreaming, decreased salivation, loss of taste, rapid pulse etc.,

In Swasakasam, there is no abdominal distention and decreased salivation.

LINE OF TREATMENT

The line of treatment of **SwasaKasam** consists of the following.

1. Kalichal Maruthuvam - To bring the dhoshas in equilibrium
2. Internal Medicine - Mainly anti-spasmodic, expectorant, to relieve the spasm and to expel the sputum.
3. Diet - To maintain tridhoshas and energy in equilibrium.
4. Prevention methods - To strengthen the muscles of respiration (Pranayama)

5. Yoga therapy - To maintain dhasa vayukkal and to improve mental and physical health.

1. Kalichal Maruthuvam (Purgation)

Patients are given laxative Nilavagai Chooranam 5 gm with hot water at the bed time on the previous day, after starting the internal medicine.

2. Administration of internal medicine

For the treatment of the disease Swasakasam, Kasa Chooranam – 1 gm and Anna Pavala Chenduram – 100mg thrice daily with honey was given before meals.

3. Diet

Siddhars advice the diet regiments for Kaba patients and they are explained below.

“கத்தரி பேய்புடல் வரை யிருபாகல் பருங்காளா கண்டகாரி
அத்திக் காய்களும் வருக்கைமாயற்றை கரையால் பீர்க்கரும் பிஞ்சுவேர்
மொய்த்த சூரணங் கதலித் தண்டுனைப் பூமுளங்கி முருக்கரும்பும்
அத்தி பூசணிக் காயருள்ளி வள்ளியுங் கபத்தோர்க் காணமாமே”

- பதார்த்த குண சிந்தாமணி

“வேளை மணத்தக்காளி மென்சீதை சக்கரவர்த்தி
பீளை வசலை சுக்கு பெண்கணங்கள் - வேளையில்
செந்தளிர் களைக்கீரை செய்வர் கபதேகர் நிதம்
வந்தளியுணத்தான் மகிழ்ந்து”

- பதார்த்த குண சிந்தாமணி

Vegetables to be added

- ❖ கத்தரி (Solanum melongena)
- ❖ பேய்புடல் (Trichosanthes cucumerina)
- ❖ அவரை (Dolichos lab-lab)
- ❖ கண்டங்கத்தரி (Solanum xanthocarpum)

- ❖ அத்தி (Ficus glomavata)
- ❖ பீர்க்கு (Luffa acutaugula)
- ❖ மாவடு (Mangifera indica)
- ❖ வாழைக்காய் (Musa paradisiaca)
- ❖ முருங்கை (Moringa tinctoria)
- ❖ சுண்டை (Solanum torvum)

Tubers to be added

- ❖ முள்ளங்கி (Raphanus sativus)
- ❖ ஈருள்ளி (Allium sativum, Allium cepa)
- ❖ இஞ்சி (Zingiber officinale)
- ❖ கருணைத்தண்டு (Amorphophallus companulatus)

Greens to be added

- ❖ மணத்தக்காளி (Solanum Nigrum)
- ❖ கரிசாலை (Eclipta alba)
- ❖ பீளை (Aerva lanata)
- ❖ வசலை (Bascella alba)
- ❖ சிறுக்கீரை (Amaranthus gangeticus)
- ❖ மணலிக்கீரை (Gisekia pharmacoides)
- ❖ பரட்டைக்கீரை (Justicia madurensis)
- ❖ புளியாரைக்கீரை (Oxalis corniculata)

Diet Restriction

Siddhars advice to avoid certain food items during diseased conditions.

They are,

“கடுகு நற்றிலத் தெண்ணெய் கூழ்பாண்டங் கடலை
வடுவ தாகிய தெங்குமா வருக்கை நற்காய
மழவி லாதவெள் ளுள்ளிகொள் புகையிலை மதுபெண்
இடறு பாகவோ டகத்தி நீக்கிடலிச் சாபத்தியம்”

- தேரையர் வெண்பா

- | | |
|----------------|----------------|
| ❖ Mustard | ❖ Gingelly oil |
| ❖ Bengal gram | ❖ Coconut |
| ❖ Mango | ❖ Jack fruit |
| ❖ Garlic | ❖ Horse gram |
| ❖ Tobacco | ❖ Alcohol |
| ❖ Bitter guard | ❖ Sesban |
| ❖ Asafoetida | |

And also, they are advised to avoid coitus

These are general diet and habitual restrictions for all diseases.

Kaba patients should restrict the followings also.

- ❖ Onion
- ❖ Jaggery
- ❖ Curd
- ❖ Butter
- ❖ Ghee
- ❖ Fish
- ❖ Dry Fish.

Prevention

1. Avoid chill and cold weather
2. Avoid working in dust, cement, cotton mills and in husks.
3. Avoid smoking
4. To sleep in phoenix mat, prevent Kaba diseases.

“சிறிற்சீசுப் பாயிற் றினமும் படுப்பவருக்
குற்றிடுமே காந்த லுடம்புவருஞ்-சுற்றியதோர்
வாயுவறும் பித்தமறு மற்றுங் கபந்தீருந்
தாயகமா மிக்குணத்தைச் சாற்று”

- அகத்தியர் குணவாகடம்

5. Advice to practice Pranayamam.

PRANAYAMAM (Breathing Exercise)

Pranayamam or breathing exercise mainly consists of Pooragam (inhalation of air by deep inspiration), Kumbagam (holding the breath as far as possible) and Resagam (exhalation of air by expiration)

During breathing exercise, the lungs filled with fresh air in its anatomical dead space also and expand well and get proper supply of oxygen by proper expansion of chest. So, Pranayama practice is one of the prevention for SwasaKasam.

By this exercise, the duration of Kumbagam is increased. This results in proper gaseous exchange which provides increased oxygen supply to the cells.

By the regular practice of Pranayamam, one can get rid mental and physical stress and enjoy pleasure. It provides good concentration and meditation. This practice also gives good appetite, strength, enthusiasm, rigor and vitality.

“நாளொன்றுக்கு இருபத்தோராயிரத்து அறுநூறு

நலமான சுவாசந்தானே முந்திருக்கும்

கோளொன்றிப் பதினாலாயிரத்து நானூறு

குவிந்த மூலாதாரத்துள் ளொடுங்கும்

பாளொன்றி யேழாயிரத்திருநூறு சுவாசம்

பாழினிற் பாய்ந்திடுமென் றறிகப்பின்னை

ஏளொன்றி யிதனையே யுட்சாதித்தால்

எப்பொழுதும் பாலரா யிருக்கலாமே”

- யூகி வைத்திய சிந்தாமணி

YOGA THERAPY

Yogasana is one of the most spiritual legacies gifted by our ancient sages. The practice of asanas strengthens the body and mind.

Asanas strengthen the muscles of respiration and diaphragm as well as regulate respiration. So, practising asanas is more helpful in asthmatic patients as supportive therapies. The following asanas are helpful in Asthma.

- ❖ Bujankasanam
- ❖ Chakrasanam
- ❖ Machasanam
- ❖ Mayurasanam
- ❖ Patha hasthasanam
- ❖ Arai machayendhirasanam
- ❖ Trikonasanam
- ❖ Savasanam.

MODERN ASPECTS

ANATOMY & PHYSIOLOGY OF RESPIRATORY

SYSTEM

The respiratory system brings air in close relationship with the mixed venous blood enabling tissue respiration by uptake of oxygen into the circulation and elimination of carbon dioxide.

The organs of the respiration are

- ❖ Nose
- ❖ Pharynx
- ❖ Larynx
- ❖ Trachea
- ❖ Two bronchi
- ❖ Bronchioles and small air passages
- ❖ Muscles of respiration - the inter costal muscles and the diaphragm

Nose and Nasal cavity

Nose is lined by ciliated columnar epithelium which contains mucus secreting goblet cells. The anterior nares or nostril are the openings from exterior into the nasal cavity. The posterior nares are the openings from nasal cavity into the pharynx.

The Nasal cavity is the first of the respiratory organs and consists of a large irregular cavity divided into two equal passages by a septum. The posterior bony part of the septum is formed by the perpendicular plate of ethmoid bone and the vomer. The roof is formed by cribriform plate of

ethmoid, sphenoid, frontal and nasal bones. The floor is formed by the roof of mouth consists of soft palate and hard palate.

The para nasal sinuses are air filled cavities in certain of the skull bones, lined by mucous membrane and communicating with the nasal cavity. The main sinuses are maxillary sinuses, frontal, sphenoidal and ethmoidal sinuses.

Respiratory Functions of Nose

The function of the nose is to begin the process by which the air is warmed, moistened and filtered. The projecting choncha increases the surface area and cause turbulence, spreading inspired air over the whole of the nasal cavity. Warming is due to immense vascularity of the mucosa. Filtering and cleaning of air occurs on hairs at the anterior nares traps layer particles. Mucous protects the underlying epithelium from irritation and prevents drying. Humidification occurs as air travels over moist mucosa and becomes saturated with water vapour. Irritation of the nasal mucosa results in sneezing a reflex action that forcibly expects an irritant.

Pharynx

Pharynx is the passage extending from the base of skull to the level of 6th cervical vertebra where it is continuous with the oesophagus. 13 cm length, 35 cm width.

Pharynx is divided into three parts. Naso Pharynx , Oro Pharynx, Laryngo Pharnx.

Naso pharynx is the nasal part of the pharynx is situated behind the nasal cavity and above the level of the soft palate. Oro pharynx extends from the level of the soft palate to the level of the upper border of the epiglottis, Laryngo pharynx extends from the upper border of the epiglottis to the lower border of the cricoid cartilage.

Functions

Passage of air and food, warming and humidifying of air, taste, hearing protection.

Larynx

The larynx is the voice box and serves as an air passages. Extends from the root of the tongue at the inlet of the larynx to the commencement of the trachea at the level of the 6th cervical vertebra. 4.3 cm length.

Functions

Production of sound, speech occurs during expiration when the sound produced by the vocal cords is manipulated by the tongue, cheeks, lips.

Protection of lower respiratory tract from the swallowed food from mouth. It is the passage for air between pharynx and trachea.

Humidifying, filtering and warming continue as the air travels through the larynx.

Trachea

Trachea is the wind pipe. It starts at the lower border of the cricoid cartilage and ends at the level of the upper border of the 5th thoracic vertebra by dividing into two bronchi right and left. 11 – 12 cm length.

Functions

Support and potency. The arrangement of the cartilage and elastic tissue events linking and obstruction of the airway on the head and the neck moves. The cartilage prevents collapse of the tube, when the internal pressure is less than intra thoracic pressure; get at the end of forced expiration.

Mucociliary escalator, this is synchronous movement of the cilia that wafts mucous with adherent particles upwards to the larynx.

Cough reflex

Nerve endings in the larynx, trachea and bronchi are sensitive to irritation that generates nerve impulse which is induced by the vagus nerve to the respiratory centre in the brain stem. The reflex motor response is deep inspiration followed with closed glottis. So the intra pleural pressures rises. Then glottis is suddenly opened with explosive out flow of air at a higher velocity. Irritates may be rapelled out of the respiratory tract.

Bronchi and Smaller Air Passage

The two bronchi are formed when the trachea divides at the level of 5th thoracic vertebra. The right bronchus is a wider, shorter tube than the left bronchus and it lies in a more vertical position. It is approximately 2.5 cm long. After entering the right lung at the hilum it divides into three branches. Each branch divides into numerous smaller branches.

The left bronchus is about 5 cm long and is narrower than the right. After entering the lungs at the hilum, it divides into two branches one for each lobe. Each lobe branch then sub divides into progressively smaller tubes with in the lung substance.

Bronchi are composed of the same tissue the trachea. They are lined ciliated columnar epithelium. The bronchus progressively subdivides into bronchioles, terminal bronchioles respiratory bronchioles, alveolar duct and finally alveoli.

Functions of air passage not involved in gas exchange

Control of air entry

The diameter of the respiratory passage may be altered by contraction and relaxation of the involuntary muscles of their walls, thus regulating the volume of air entering the lungs. These changes are controlled by the autonomic nerve supply. Parasympathetic stimulation causes constriction and sympathetic stimulation causes dilation.

The following functions continue as in the upper airways.

- ❖ Warming and humidifying
- ❖ Support and potency
- ❖ Removal of particulate matter
- ❖ Cough reflex

Respiratory Bronchioles and Alveoli

Lobules are blind ends of the respiratory tract, distal to the terminal bronchus consist of respiratory bronchioles, alveolar duct and alveoli. The walls gradually thinner until muscle and connective tissue fade out leaving a single layer of simple squamous epithelial cells in the alveolar duct and alveoli. These distal respiratory passages are supported by a loose network of capillaries. The exchange of gases during respiration takes place across two membranes - alveolar and capillary membrane.

Interspersed between the squamous cells are other cells that secrete surfactant, a phospholipid, fluid which prevents the alveoli from drying out. In addition, surfactant reduces the surface tension and prevents alveolar walls collapsing during expiration.

Functions of respiratory bronchioles and alveoli

1. External respiration
2. Defence against microbes

Cells in connective tissue protect against infection and inhaled foreign particles not trapped by mucous. Lymphocytes and plasma cells produce antibodies in the presence of antigen and macrophages and poly morpho nuclear lymphocytes are phagocytic. These cells are most active in the distal air passages where ciliated epithelium has to be replaced by flattened cells.

Warming and humidifying continue as in the upper airways. Inhalation of dry or inadequately humidified air over a period of time causes irritation of the mucosa and facilitates the establishment of pathogenic microbes.

Lungs

Lungs are paired organs of respiration. They are situated one on each side of the mediastinum within the thoracic cavity. Each lung resembles a half cone. It has an apex, a base, medial surface and costal surface.

Right lung is broader than the left lung and weighs 220g, and is divided into three lobes, whereas the left lung weighs 200g and is divided into two lobes. The apex is rounded and rises into the roof of the neck about 25mm above the level of the middle third of the clavicle. The base is concave and semilunar in shape and is closely associated with the thoracic surface of the diaphragm.

The costal surface is convex and is closely associated with the costal cartilages, the ribs and the intercostal muscles. The medial surface is concave and has a roughly triangular shaped area, called hilum at the level of 5th, 6th, 7th thoracic vertebra. Structures that enter and leave at the hilum are 1 bronchus, 1 pulmonary artery, 2 pulmonary veins, 1 bronchial artery, 1 bronchial vein, lymph vessels, parasympathetic and sympathetic nerves. The area between the lungs is the mediastinum. It is occupied by heart, great vessels, trachea, right and left bronchi, oesophagus, lymph nodes, lymph vessels and nerves.

Pleura and Pleural Cavity

Each lung is covered by the pleural cavity. The pleura consist of a closed sac of serous membrane, which contains a small amount of serous fluid. The visceral pleura is adherent to the lungs covering each lobe and passes into the fissures which separate them. The parietal pleura is adherent to the inside of the chest wall and the thoracic surface of the diaphragm and is continuous with the visceral pleura at the hilum.

RESPIRATION

Inflation and deflation of the lungs ensues that regular exchange of gases takes place between alveoli and external air. This is dependent upon the arrangement of pleura and the contraction and relaxation of muscles of respiration and the elastic connective tissue.

Muscles of Respiration

The expansion of the chest during inspiration occurs partly voluntary and partly involuntary. The muscles of normal quiet breathing are the inter costal muscles and the diaphragm. During difficult breathing they are assisted by the muscles of the neck, shoulder and abdomen.

Cycles of Respiration

This occurs 12-15 times per minute and consists of three phases.

- ❖ Inspiration
- ❖ Expiration
- ❖ Pause

Inspiration

The capacity of the thoracic cavity is increased by simultaneous contraction of the inter costal muscles and the diaphragm. The parietal pleura move with the walls of thorax and the diaphragm. This reduces the pressure in the pleural cavity to the level considerably lower than the atmospheric pressure. The visceral pleura follow the parietal pleura. During the process, the lungs are stretched, the pressure within the alveoli and the air passage reduced drawing air into the lungs in an attempt to equalize the atmospheric and alveolar air pressure.

The process of inspiration is active as it requires expenditure of energy for muscle contraction. The negative pressure created in the thoracic cavity aids venous return to the heart and is known as respiratory pump.

Expiration

Relaxation of inter costal muscles and the diaphragm results in the downward and inward movement of the rib cage and the elastic recoil of the lungs. As this occurs, the pressure of the gases inside the thorax exceeds the atmospheric pressure and therefore air is expelled from the respiratory tract. The lungs still contain some air and are prevented from complete collapse by the intact pleura. The process is passive as it does not require the expenditure of energy.

After expiration there is a pause, before the next cycle begins.

Physiology Variables Affects Respiration

Elasticity

Loss of elasticity of the connective tissue in the lungs necessitates forced expiration and increased effort of inspiration.

Compliance

This is the measure of distensibility of the lungs, I. e., the effort required to inflate the alveoli when compliance is low, the effort needed to inflate the lungs is greater than normal e.g. in some diseases where elasticity is reduced or when surfactant is present insufficiently.

Air flow resistance

When this is increased e.g. in broncho constriction, more respiratory effort is required to inflate the lungs.

Lung volumes and capacity

In normal quiet breathing there are about 15 complete respiratory cycles per minute. The lungs and air passages are never empty and as the exchange of

gases take place only across the wall of the alveolar ducts and alveolar. The remaining capacity of the respiratory passages is called the anatomical dead space (about 150ml).

Tidal Volume

It is the amount of air which passes into and out of the lungs during each of quite breathing about 500ml.

Inspiratory Reserve Volume

It is the extra volume of air that can be inhaled into the lungs during maximal inspiration.

Inspiratory Capacity

It is the sum of Tidal Volume and Inspiratory Reserve Volume.

Functional Residual Capacity

It is the amount of air remaining in the air passages and alveoli at the end of quiet respiration. The functional residual volume also prevents the collapse of the alveoli on expiration.

Expiratory Reserve Volume

It is the largest volume of air which can be expelled from the lungs during maximal expiration.

Vital Capacity (VC)

It is the maximum volume of air which can be moved into and out of the lungs.

Residual Volume

It cannot be measured directly but, it is the volume of air remaining in the lungs after forced expiration.

$$VC = \text{Tidal Volume} + \text{IRV} + \text{ERV}.$$

Alveolar Ventilation

This is the volume of air that moves into and out of the alveoli per minute. It is the tidal volume minus the anatomical dead space, multiplied by the respiratory rate.

$$\begin{aligned}\text{Alveolar ventilation} &= (\text{TV}-\text{anatomical dead space}) \text{ respiratory rate} \\ &= (500-150) \text{ ml} \times 15 \text{ per minute} \\ &= 5.25 \text{ liters / minute.}\end{aligned}$$

Lungs function tests are carried out to determine respiratory function and are based on the parameters outlined above.

External Respiration

This is the exchange between alveoli and blood. Total area of gas exchange in the lungs is 70-80 square meters. CO₂ diffuses from venous blood along the concentration gradient into the alveoli until equilibrium with alveolar air is reached. By the same process O₂ diffuses from alveoli to the blood.

Internal Respiration

This is the exchange of air between the tissue and blood. When there is difference in partial pressures, oxygen diffuses outward from the blood to extracellular fluid then into the cell walls. The process involved is diffusion.

Control of Respiration

Control of respiration is normally involuntary. Voluntary control is exerted during activities such as speaking, singing but is overridden if homeostasis of arterial PO₂ and PCO₂ is threatened i.e. if this is high arterial PCO₂ or low arterial PO₂.

THE RESPIRATORY CENTRE

This is formed by group of nerve cells that control the rate and depth of respiration. They are situated in

- ❖ Brainstem
- ❖ Medulla oblongata
- ❖ Pons varoli

In the medulla there are inspiratory neurons and expiratory neurons. Neurons in the pneumotoxic and apneutic centre situated in the pons influence the inspiratory and expiratory neurons of the medulla.

Motor impulses leaving the respiratory centre pass in the phrenic nerves and inter costal nerves to the diaphragm and inter costal nerves.

Chemoreceptor

These are the receptors that respond to the changes in PO_2 and PCO_2 . They are located centrally and peripherally.

Central receptor - present on the surface of medulla oblongata and bathed in CSF. When PCO_2 is raised even slightly, the central receptors respond by stimulating respiratory centre, by increasing ventilation and reducing PCO_2 . The sensitivity to raised PCO_2 is the most important factor in maintaining hemostasis of blood gases in health.

Peripheral chemoreceptors are situated in the arch of aorta and in the carotid bodies.

An increase in H^+ concentration stimulates the peripheral chemoreceptors resulting in increased ventilation, increased CO_2 excretion and reduced P^H . Other factors influencing respiration are,

- ❖ Speech, singing
- ❖ Emotional displays
- ❖ Drugs e.g. - sedatives, alcohol
- ❖ Sleep

Temperature influences breathing. In fever, respiration is increased due to increased metabolic rate while in hypothermia it is decreased. It is depressed as in metabolism, temporary changes in respiration occur in swallowing, sneezing and coughing.

BRONCHIAL ASTHMA

Bronchial Asthma is a disease of airways that is characterised by increased responsiveness of the tracheo bronchial tree to a variety of stimuli, resulting in widespread spasmodic narrowing of the air passages which may be relieved spontaneously or by therapy.

Asthma is an episodic disease manifested clinically by paroxysms of dyspnea, cough, polyphonic wheeze. However, a severe and unremitting form of the disease termed status asthmaticus may prove fatal.

Prevalence

Asthma is common and prevalent world wide. It occurs at all ages but nearly 50% of cases develop before the age of 10 years. In adults, both sexes are affected equally, but in children there is 2:1 male – female ratio.

Aetiology

From the aetiological point of view, asthma is heterogenous disease. It is useful for epidemiological and clinical purposes to classify asthma by the principle stimuli. There are two types of asthma.

- ❖ Early onset asthma (atopic, allergic, extrinsic)
- ❖ Late onset asthma (Non-atopic, Idiosyncratic, Intrinsic)

Atopic Asthma

This is the most common type of asthma usually begins in childhood. The disease is triggered by environmental antigens, such as dust, pollens, animals dander, fungal spores and food. A positive family history of atopy is common, and asthmatic attacks are often preceded by allergic rhinitis, urticaria or eczema. Serum IgE levels are usually elevated. A skin test with antigen results in an immediate wheal and flare reaction, a classic examples of type I - IgE mediated hypersensitivity reaction.

Non – Atopic Asthma

This type of asthma develops later in adult life with negative personal (or) Family history of allergy, negative skin test and normal serum levels of IgE. Most of these patients develop typical symptom-complex after an upper respiratory tract infection by viruses (e.g. rhinovirus, para influenza virus). Associated nasal polyp and chronic bronchitis are commonly present. About 10% of patients become hypersensitive to drugs, most notably to small doses of aspirin.

Pathogenesis of Asthma

The common denominator underlying the asthmatic diathesis is a nonspecific hyper irritability of tracheo-bronchial tree. When airway reactivity is high, symptoms are more severe and persistent and the magnitude diurnal fluctuation in lung functions is greater. The patients tend to awaken at night or in the early morning with breathlessness.

In both normal and asthmatic patients, air reactivity rises following viral infections of the respiratory tract and exposure to oxidants such as ozone and nitrogendioxide. Allergen can cause airway responsiveness to rise within minutes and remain elevated for weeks.

A number of causes have been postulated for the increased airway activity of asthma, but the basic mechanism remains unknown. The most popular hypothesis at present is that of airway inflammation. Increased numbers of mast cell, epithelial cells, neutrophils, eosinophils and lymphocytes have been found in the broncho alveolar lavage fluid of patients with asthma & have number of mediators.

The airway can be oedematous and infiltrated with eosinophils, neutrophils and lymphocytes with or without the swelling of the epithelial basement membrane. There may be glandular hypertrophy. The most obvious

finding is a generalised increase in cellularity associated with an elevated capillary density.

Although, the translation of this histological observation into the disease is still incomplete, it is widely believed that the physiological and clinical features of asthma derived from interaction among the residence and infiltrating inflammatory cells in the airway and the surface epithelium. The cells that play more important role are mast cells, eosinophils, macrophages, neutrophils and lymphocytes. The mediators released are histamine, bradykinin, the leukotrienes C, D, & E, platelet activating factor (PAF) and prostaglandins (PGs) E_2 , F_{2a} and D_2 produce an intense, immediate inflammatory reaction involving broncho constriction, vascular congestion and oedema formation. In addition to their ability to evoke prolonged constriction of airway smooth muscles and mucosal edema, the leukotrienes may also account for some of other patho physiological features of asthma such as increased mucous production and impaired mucociliary transport.

Chemotactic factors elaborated bring eosinophil, platelets and polymorphic nuclear leukocytes to the site of reaction. These infiltrating cells and resident macrophages and airway epithelial cells themselves potentially are an additional source of mediators to enhance immediate and the cellular phase.

Like mast cells in the early reaction the eosinophils play an important role in the infiltrative components. The granular protein in thin cell major basic protein and the eosinophilic cationic protein are capable of destroying the airway epithelium, which then sloughed into the bronchial lumen in the form of creak bodies. Besides resulting in a loss of barrier and secretory function, such damage elicits the production of chemostatic cytokines leading to further inflammation. In theory it also can expose sensory nerve endings, thus irritating

neurogenic inflammatory pathways. That in turn, converts a primary local event into a generalised reaction via a reflex mechanism.

T lymphocytes also appear to be important in the inflammatory response. These cells are present in increased number in asthmatic airways and produce cytokines that activate cell-mediated immunity as well as humoral (IgE) immune response.

Further more, Th₁, Th₂ lymphocyte subtype have functions that may influence the asthmatic response. The Th₁ Cytokines interleukin (IL-2) and interferons (IFN) γ , can promote the growth and differentiation of β cells and activation of macrophages respectively.

T_{H2} cytokine IL-4 and IL-5 stimulate β cell growth and immunoglobulin secretion and IL-5 promote eosinophilic proliferation, differentiation and the activation. It can also facilitate granule release from basophils.

The stimuli that interact with airway responsiveness and incite acute episodes of asthma can be grouped into nine major categories - allergenic, pharmacological, environmental, occupational, infections, and exercise - related and emotional stress, food and drink, smoking.

1. Allergens

Allergic asthma is dependent on IgE response controlled by T and B lymphocytes and activated by the interaction of antigen with mast cells-bound IgE molecule. Most of the allergens are airborne and to induce a state of sensitivity, they must be reasonably abundant for considerable period of time.

Allergic asthma is frequently seasonal and it is most after observed in children and young adults. A non-seasonal form may result from allergy to feathers, animal dander, dust mites, molds and other antigens that are present continuously in the environment.

2. Pharmacological stimuli

The drugs most commonly associated with the induction of acute episodes of asthma are aspirin, colouring agents - tartazine, β - adrenergic antagonists, sulfating agent.

Aspirin - sensitive syndrome affects adults through seen in childhood. The problem usually begins with perennial vasomotor rhinitis that is followed by a hyper plastic rhino sinusitis with nasal polyps, progressing to asthma. Indomethacin, fenoprofen, naprofen, zonepirae sodium, ibuprofen, mefenamic acid and phenylbutazone are particularly important.

β - Adrenergic antagonist regularly obstructs the airway in asthmatics. In fact, the local use of β - blockers in the eye for the treatment of glaucoma has been associated with worsening asthma.

Sulfating agents can produce acute airway obstruction in sensitized individuals. Exposure usually follows ingestion of food and beverages containing these compounds. e.g. - salads, fresh fruit, potatoes, shell fish & wine.

3. Environment and Air pollution

Environmental causes of asthma are usually related to climatic conditions that promote the concentration of atmospheric pollutants and antigens. These conditions tend to develop heavily industrial or densely populated urban areas and frequently associated with thermal inversion or other situation that cause stagnant air masses. The air pollutants known to have this effect are ozone, nitrogen dioxide & sulphur dioxide.

4. Occupational factors

Occupation - related asthma is a significant health problem and acute and chronic airway obstructions have been reported to follow exposure to a large number of compounds used in many types of industrial process.

Broncho constriction can result from working with or being exposed to metal salts, wood and vegetable dust, husk of grains, flour, castor bean, gum acacia, karay gum, tragacanth, pharmaceutical agents e.g. antibiotics, piperazine and cimetidine, industrial chemicals and plastics, biological enzymes, laundry detergents and pancreatic enzymes, animal & insect dusts, serum and secretions.

There seems to be three underlying mechanisms

1. In some cases, the offending agent results in formation of significant IgE.
2. Substances cause direct liberations of broncho constrictor substances.
3. Substances cause direct or reflex stimulation of the airway of latent or frank asthmatics.

5. Infection

Respiratory infections are the most common of the stimuli that evoke exacerbations of asthma. In young children, the most important infectious agents are respiratory syncytial virus and para influenza virus. In older children and adults rhino virus and influenza virus predominate as pathogens. *Streptococcus pneumoniae*, *H.influenza* and viruses are responsible for infection.

Simple colonization of the tracheo bronchial tree is insufficient to evoke acute attacks & attack of asthma occurs only when the symptoms of an ongoing respiratory tract infection are having been present. The mechanism by which viruses induce exacerbations of asthma may be related to the production of T lymphocyte derived cytokines that potentiate the infiltration of inflammatory cell into already susceptible airways.

6. Exercise

Exercise is one of the most common precipitating factors in acute episodes of asthma. Exercise probably invokes broncho spasm to some extent in every asthmatic patient, and in some it is the only trigger that produces symptoms. The mechanisms, by which exercise produce obstruction, may be related to a thermally produced hyperthermia and engorgement of the microvasculature of the bronchial wall and doesn't appear to involve smooth muscle contraction.

7. Emotional Stress

Psychological factors can interact with the asthmatic diathesis to worsen or ameliorate disease process. Changes in airway caliber seem to be mediated through modification of nasal efferent activity, but endorphins also may play a role.

8. Food and Drink

Atopic asthmatics may occasionally notice that their symptoms are provoked by certain foods or drinks and it is worth enquiring of all asthmatic patients whether they have notices such as association.

The food most frequently suspected the milk, eggs, fish, cereals, nuts and chocolates. Preservatives such as benzoates, sodium nitrite, and sodium metabisulphite, anti oxidants, dyes such as tartarazine, flavorings may be found in many food and may provoke asthma. Red wines contain a number of congeners which give them their distinctive flavors but which also may provoke attacks of asthma.

9. Smoking

Smokers appear to be at greater risk of developing asthma and have a higher prevalence of hyper-reactivity. Children of smokers also seem to have an increased risk of developing wheeze.

PATHOLOGY

In patient who died of acute asthma the most striking feature of lung at mesopry is their gross over distension and failure to collapse when the pleural cavities are opened. When the lung is cut, numerous geletinous plugs of exudates are present in most of bronchical branches down to the terminal bronchioles. Histological examination shows the hypertrophy of the bronchial smooth muscle, hyperplasia of mucosal, sub mucosal vessels, mucosal oedema denudation of the surface epithelium, pronounced thickening of the basement membrane and eosinophilic infiltration in the bronchial wall. In smaller proportions of asthmatics that die, the eosinophilic infiltration is replaced by neutrophils and mucous plugging is conspicuously absent.

PATHO PHYSIOLOGY

The patho physiological hallmark of asthma is a reduction is airway diameter brought about by contraction of smooth muscles, vascular changes, congestion, oedema of the bronchial wall and thick tenacious secretions. The net result is an increase in airway resistance, a decrease in forced expiratory volumes and flow rates, hyperinflation of the lungs and thorax, increased work of breathing alternation in respiratory muscle function, changes in elastic recoil, abnormal distribution in both ventilation and pulmonary blood flow with mismatched ratios and alternations to the arterial gas concentrations. Thus, although asthma is considered to be primarily a disease of airways, virtually all aspects of pulmonary functions are compromised during acute attacks. In addition in very symptomatic patients the ECG frequently shows right ventricular hypertrophy and pulmonary hypertension. When a patient is present for therapy, his or her forced vital capacity tends to be < 50% of normal. The forced expiratory volume averages 30% of less of predicted, while maximum

and minimum mid-expiratory flow rate are reduced to 20% or less of the expected value.

In acutely ill patient, RV frequently approaches 400% of the normal, while functional residual capacity doubles. The patient tends to report that the attack has ended clinically, when RV has fallen to 200% of its predicted value and FEV has reached 50% of the predicted level.

Hypoxia is a universal finding during acute exacerbations, but frank ventilatory failure is relatively uncommon, most asthmatics have hypoapnea and a respiratory alkalosis. In acutely ill patients the finding of a normal arterial CO₂ tends to be associated with quite severe levels of obstruction.

Cyanosis is a very late sign. Likewise, signs attributes to CO₂ retention, such as sweating, tachycardia, wide pulse pressure or to acidosis, such as hyperapnea or H⁺ excess in individual patient, because they are too frequently seen in anxious patients with more moderate disease.

CLINICAL FEATURES

The symptoms of asthma consist of a triad of dyspnoea, cough and wheezing the last often being regarded as the sine quo none. In its most difficult form, asthma is an episodic disease and all the three symptoms co-exist. At the onset of attack patient experiences a sense of constriction in the chest often with a non-productive cough. Respiration becomes audibly harsh, wheezing in both phases of respirations becomes predominant, expiration is prolonged patient frequently has tachypnoea, tachycardia and mid-systolic hypertension. The lungs rapidly become over-inflated and antero posterior diameter is increased. If the attack is severe or prolonged, there may be loss of adventitious breath sounds and wheezing becomes very high pitched. Further the accessory muscles, which become visible active and a paradoxical pulse often develops. These two signs have been found to be extremely valuable

indicating the severity of the obstruction. Pulmonary function tends to be significantly more impaired. The development of paradoxical pulse depends on the generalisation of large negative intra thoracic pressure. Thus, if the patient's breathing is shallow, these signs and / or the use of accessory muscles could be absent even though obstruction is quite severe.

The end of the episode is frequently marked by a cough, that produce stringy mucus which often takes the form of casts of the distal airway crushman's spirals and when examined microscopically often shown eosinophils and Charcot - leydian crystals. In extreme situations, wheezing may lessen markedly and even disappear cough may become extremely ineffective and the patient may begin a gasping type of respiratory pattern. These findings imply extensive mucous plugging and impeding suffocation. Ventilator assistance is required. Atelectasis due to inspissated secretion occasionally occurs with asthmatic attack.

Less typically, a patient with asthma may complain of intermittent episode of nonproductive cough or excretional dyspnoea. Unlike other asthmatics, when the patients are examined during symptomatic period, they tend to have normal breathing, but may have wheeze after repeated forced expiration and or may have ventilatory impairments when tested in the laboratory. In the absence of these signs a broncho - provocation test may be required to make the diagnosis.

1. Acute Severe Asthma (Status Asthmaticus)

1. It is a medical emergency. Patient is hypoxic and cyanosed due to severe bronchospasm.
2. Severe dysphoea, unproductive cough, patient adopts an upright position fixing the shoulder girdle to assist the accessory muscles of respiration.
3. It is characterized by tachycardia (pulse rate > 120) tachypnoea (respiratory rate > 30/ minute) sweating, pulsus paradoxus, altered level of consciousness and an inspiration, expiration ratio of 1: 3 or 1: 4.
4. The presence of a silent chest and bradycardia in such patients is an ominous sign.

ACUTE SEVERE ASTHMA – GRADE:

- | | | |
|---------------------------|---|---|
| Grade₁A | - | Able to carry out house work or job with moderate difficulty.
Sleep occasionally disturbed. |
| Grade₁B | - | Only able to carry out house work or job with great difficulty.
Sleep frequently disturbed. |
| Grade₂A | - | Continued to chair (or) bed, but also to get up with moderate difficulty. Sleep disturbed, with little or no relief from inhaler. |
| Grade₂B | - | Confined to chair or bed and only able to get up with great difficulty unable to sleep. Pulse over 120/min. |
| Grade 3 | - | Totally confined to chair or bed. No sleep. No relief from inhaler. Pulse over 120/min. |
| Grade 4 | - | Immobilized and completely exhausted. |

2. Nocturnal Asthma

Nocturnal asthma is defined as an overnight fall of more than 20% in the FEV₁ or PEF. It may be the sole manifestation of asthma. This is presumed to be due to

- a. Early morning fall in circulating adrenaline
- b. Overnight changes in vagal tone (increased vagal tone in early morning)
- c. Airway cooling at night.
- d. Circadian changes in plasma cortisol concentration (mid night to early morning fall in cortisol level).

3. Gastric Asthma

Worsening of asthma after meals or dyspnoea occurring only after meals is due to gastro-oesophageal reflex.

4. Cough Variant Asthma

Cough may be the dominant symptoms and the lack of wheeze (or) breathlessness.

DIFFERENTIAL DIAGNOSIS

The differentiation of asthma from other disease associated with dyspnoea, wheezing is usually not difficult, particularly when the patient is seen during acute attacks.

The physical sign and symptoms listed above and the history of periodic attacks are quick characteristic. A personal history or family history of allergic disease such as eczema, rhinitis or urticaria is valuable contributory evidence. An extremely common feature of asthma is nocturnal awakening with dyspnoea and / or wheezing. In fact, the phenomenon is so prevalent that, its absence raises doubts about the diagnosis.

Upper airway obstruction by tumour or laryngeal oedema can occasionally confuse with asthma. Typically a patient with such a condition will present with stridor and harsh breathing sound that can be localised to the area of trachea. Diffuse wheezing through out the lung field is usually absent. However, differentiation can sometimes be difficult and indirect laryngoscope or bronchoscope may be required. Asthma like symptom has been described in patients with glottis dysfunction. These persons narrow their glottis during inspiration and expiration.

Persistent wheeze localised to one area on the chest is associated with paroxysms of coughing indicates endobronchial diseases, such as foreign body aspiration a neoplasm or bronchial stenosis.

The signs and symptoms of acute left ventricular failure occasionally mimics asthma, but the finding of moist basilar rales, gallop rhythm, blood-stringed sputum and other signs of heart failure allow appropriate diagnosis to be reached. Recurrent episodes of bronchospasm can occur with carcinoid tumour, recurrent pulmonary emboli and chronic bronchitis.

In chronic bronchitis, there are no true symptom-free periods and one can usually obtain a history of chronic cough and sputum production as a background upon which acute attack wheezing are superimposed. Frequently patients with this condition will present the episode of breathlessness particularly on exertion and they sometimes wheeze.

NO	FACTORS	CARDIAC ASTHMA	BRONCHIAL ASTHMA
1	Past history	Hypertension, aortic or coronary disease	Previous attacks of asthma or other allergic conditions in patients of other members of the family
2	Age	Onset usually after 50 yrs	Any age
3	Precipitating factors	May be precipitated by exertion or acute myocardial infarction or hypertension	Trigger factors may be infected non specific irritants, external, allergies, exercise of emotional factors,
4	Symptoms: a. Cough	Cough and dyspnoea, cough associated with watery expectoration which increases in intensity towards end of attacks.	Starts with dyspnoea, expectoration of small sticky sputum, paroxysm of wheezes when cough becomes profuse
	b. Wheezing	Rare	Usual
	c. Sweating	Prominent	Rare, unless status asthmaticus
5	Signs:		
	A. Inspection i. Accessory muscles of respiration ii. Shape of the chest	Not active Normal	Active Emphysematous
	b. Palpation	Heart offers enlarged having palpable apex beat	Heart not enlarged, if long standing disease, right ventricular enlargement
	c. Auscultation	S ₂ may be loud. Left ventricular gallop. Expiration not unduly prolonged, rales more than bronchitis, signs in early stage at base of the lungs, gradually ascending up with progress of the attack	Normal A ₂ sound, right ventricular gallop is later feature of severe bronchial asthma. Expiration markedly prolonged rhonchi more than rales. Signs diffuse all over the lungs.

	d. Pulse	Full and bounding	Feeble and rapid
	e. B.P	Usually elevated	Normal or low
	f. Signs of underlying disease	Hypertension or coronary disease	No evident of cardio vascular disease
	h. Urine	Generally clear, then may be mild albuminuria.	Clear
6.	Investigation: Eosinophil	None	Common

NO	FACTORS	TROPICAL EOSINOPHILIA	BRONCHIAL ASTHMA
1	Age	Any age	Usually starts before 3 yrs of age
2	Duration of symptom	Short duration	Long duration
3	Cough and dyspnoea	Dyspnoea more than cough, breathlessness particularly after cough	Paroxysmal cough more than dyspnoea.
4	Fever	Common	Rare
5	Loss of weight	Fairly common	Seldom
6	Auscultatory signs	Disproportion between cough and breathlessness	Compatible with degree of cough and breathlessness
7	Blood	Leukocytosis, Eosinophilia	Normal WBC count eosinophilia 8 to 15%

NO	FACTORS	PULMONARY TUBERCULOSIS	BRONCHIAL ASTHMA
1	Age	Generally aged persons	Usually starts before 3 yrs of age
2	History	History of chronic cough	History of previous attacks.
3	Duration of symptoms	May last longer	May last up to old age
4	Time of onset	-	Early onset
5	Mode of onset	May be precipitated lay infection	May be precipitated by allergy
6	Loss of weight	Common	Seldom
7	Symptoms:		
	i. Fever	Various extent	Rare
	ii. Cough	Frequent, sharp, short, may be dry in the early stages, later it is persistent with copious, purulent, expectoration dyspnoea is later feature.	Paroxysmal cough more than dyspnoea
	iii. Wheezing	localised wheezing due to bronchial narrowing by tuberculous lymph nodes	wheezing present all over the field
	iv. Sweating	Especially during night	rare, unless in asthmatics
	v. Haemoptysis	Early stage blood stained sputum	Nothing relevant
8	Inspection	Affected side of chest flattered with displaced, apex impulse to the side of lesion, clubbing of fingers, present	No fluttering of the chest, apical impulse in position.
9.	Palpation	Movement of chest in affected side, vocal fremitus diminished, increased in consolidation, lymphadenopathy is noted.	In longstanding cases, right ventricular enlargement.

10	Percussion	Dull note in the apex, others impaired	Normal
11	Auscultation	Breath sounds bronchial early wheezing, late crackling rales, diminished vocal resonance in early and increased in later conditions	Prolonged expiration wheezing rhonchi heard all over the field
	a. Pulse	Increased or low	Normal or low
	b. B.P.	Low	Normal or low
	c. Signs of underlying disease	-	No evidence of cardio vascular disease
	d. Sputum	Hard, thick, tenacious sputum, positive in culture.	Sticky pellets.
	e. Blood	Lymphocytosis, raised ESR	Eosinophils, ESR normal.

DIAGNOSIS AND INVESTIGATION

An account of episode wheeze, breathlessness interpreted with period of normality is sufficient evidence to suspect asthma and further evidence comes from a history of marked variability, attacks in small hours of the night, provocation by strong exercise and allergens and paroxysmal cough, productive small amount of sticky sputum.

CONFIRMATION OF THE DIAGNOSIS

Lung function tests

The physiological function of the lung can be accurately by lung functional testing. Many sophisticated tests are possible but even the simplest equipment can produce useful information. It is of particular value to perform lung function tests in order to give an objective assessment of lung performance. The commonest reason for performing a lung function test is to reach a diagnosis. In addition, these tests can be most useful for following the progress of a disease and for testing objectively the effects of treatment.

Spirometry

There are many types of spirometry available and all record similar information. The traces are obtained by a deep inspiration followed by the fastest possible and maximal expiration.

The normal forced vital capacity (FVC) is fully exhaled in less than 3 seconds more than three quarter is exhaled in the first second. In addition to measuring the vital capacity in liters, the volume expired in the first second or the first expiratory volume at 1 second (FEV_1) can be measured and the ratio of $FEV_1 : FVC$ can be calculated. The results are compared with predicted values based on age height and ethnic group. FEV_1/FVC ratio is normally greater than 70%.

If diffuse airflow obstruction is present, the rate at which the air can be exhaled is diminished throughout expiration. The length of expiration is prolonged and FEV_1 is much reduced. This is an obstructive pattern and is seen in as time, chronic bronchitis and emphysema. In patients with asthma this obstruction may be reversible by treatment with bronchodilators or even corticosteroids.

In a restrictive pattern, there is a reduced lung volume, perhaps as a result of pulmonary fibrosis or chest wall deformity but there is no obstruction to airflow and FEV_1 is normal.

Peak Expiratory Flow Meter

It is a popular instrument for assessing airflow obstruction there is a cheap, simpler version called the mini peak flow meter which is suitable for use at home by individual patients. These machines measure the maximal rate of flow which is achieved during a forced expiration and most healthy people will achieve values of greater than 400 liters/min. Patients with lung fibrosis and restriction changes on the spirogram may also have normal expiratory flow

rates so the meter is not suitable for assessment of their disability. Patients with airflow obstruction will have reduced flow rates, with values below 200 liters/min being very significant and those below 100 liters/min extremely severe.

Flow Volume Curves

The plotting of flow versus volume during both maximal expiratory and inspiratory manoeuvres is of major help in differentiating central airflow obstruction (leading to stridor) from diffuse airflow obstruction as seen in COPD and asthma.

Lung Volumes

Measurement of total lung capacity and residual volume is best performed using a whole body plethysmograph, but can be measured by a helium dilution method. In general, restrictive defects lead to reduced values, and obstructive defects to increased values.

Measurement of Diffusing Capacity

The diffusing capacity (DLCO) is a measure of the lung's ability to transfer gas from alveoli to blood. The test utilized uptake of carbon monoxide from a single breath of 0.3% mixture in air; this gas is chosen because it combines rapidly with hemoglobin and provides a true estimate of diffusion across the alveolar capillary membrane. The diffusing capacity is reduced in patients with disease principally affecting alveoli such as fibrosing alveolitis or emphysema. The transfer coefficient (KCO) is a measure of diffusing capacity expressed per volume of ventilated lung during the single breath test and is useful to confirm that a low DLCO is due to alveolar disease rather than maldistribution of ventilation. High values of DLCO may be seen in alveolar hemorrhage.

Arterial Blood Gases and Oximetry

Measurement of hydrogen ion concentration. PaO_2 and PaCO_2 , and derived bicarbonate of arterial blood are essential in assessing the degree and type of respiratory failure and for measuring overall acid-base status. Use of a pulse oximeter allows a non-invasive continuous method of assessing oxygen saturation in patients who require continuous monitoring in order to assess hypoxaemia and its response to therapy, including supplemental oxygen.

Exercise Tests

Formal exercise testing with measurement of metabolic gas exchange and respiratory and cardiac response using cycle ergometry or treadmill exercise is useful in providing a detailed analysis of both pulmonary and cardiac function in the breathless patients. Exercise challenge with measurement of spirometry before and after can also be helpful in demonstrating exercise – induced asthma. Finally, the 6 minute walk test or ‘shuttle’ test can provide a simple but objective assessment of disability and response to treatment.

Skin Hypersensitivity Tests

A prick is made in the skin with a fine needle through a drop of an aqueous extract of the substance to be tested. A positive reaction is indicated by the development of a wheal and flare, which begins to appear within few minutes. Tests are usually performed with a group of common allergens known to cause bronchial asthma. It is seldom possible with these tests to identify the one particular allergens as the causes of asthma are an individual patient and their chief value is to distinguish atopic from non-atopic subjects.

Physical Signs of The Chest

During an attack of asthma, the following signs are detectable.

Respiratory rate is increased with the use of accessory muscles of respiration. Hyper- resonant percussion note over the lungs. Breath sounds are vesicular in character with prolonged respiration. Numerous high pitched polyphonic expiratory and inspiratory rhonchi are audible.

During very severe attacks the airflow may be insufficient to produce rhonchi. This results in a 'silent chest' which is an ominous sign. In between attacks the chest is clear and no abnormal physical signs may be detectable. Chronic asthmatics usually have some scattered rhonchi persisting always in their chest.

Radiology Examination

In an acute attack of asthma, the lungs appear hyper inflated. Between episodes the chest x-ray is usually normal. In long standing cases, the appearance may be indistinguishable from hyper-inflation caused by emphysema and a lateral view may demonstrate a 'pigeon chest' deformity. Occasionally when a large bronchus is obstructed by tenacious mucus, there is opacity caused by lobar or segmental collapse.

A chest x-ray should be performed if possible in all patients with severe acute asthma to exclude pneumothorax a rare but potentially fatal complication of the pulmonary hyper-inflation produced by severe airflow obstruction in asthma. The chest x-ray shows mediastinal and subcutaneous emphysema in very severe disease.

Sputum Examination

Sputum eosinophilia is a useful indication of an asthmatic type of airway reaction. Stained sections of sputum fixed in alcohol or formalin is probably a severe indication of asthma than a sputum eosinophil count. This is useful for

the demonstration of *Aspergillus fumigatus*. Eosinophils are a prominent feature of the inflammatory exudates within the airway lumen lies a thick tenacious mucus which under the microscope is seen to contain strips of desquamated epithelial cells (Curschman's spirals) eosinophils, isolated metaplastic epithelial cells (Creola bodies) & crystalline materials consisting largely of major basic protein derived from eosinophilic granules. (Charcot - leydon crystals).

COMPLICATIONS

Mortality is uncommon in asthma but a severe attack may result in respiratory failure and death.

This is more in 'status asthmaticus'. Other complications include frequent respiratory infection, pulmonary collapse due to obstruction by viscid secretions, pneumothorax, and emphysema and cough fracture (fracture of ribs due to violent coughing), children with asthma may show retardation of growth, especially if treated with corticosteroid on a long term basis. Long standing bronchial asthma, punctuated with frequent expiratory infections may lead to emphysema and chronic cor pulmonale.

Pregnancy

Three problems

- 1 The effects of asthma on pregnant women.
- 2 The effects of drugs on the child.
- 3 The chances of having an asthmatic child.

PROGNOSIS

The prognosis of the individual attack is good, except in severe acute asthma, when there is occasionally a fatal outcome, especially if treatment is inadequate or delayed. Spontaneous remission is fairly common in episodic asthma, particularly in children, but rare in chronic asthma, which can lead to

irreversible airflow obstruction. Seasonal fluctuation can occur in both types of asthma. Atopic subject with episodic asthma are usually worse in the summer, when they are more heavily exposed to antigens, while chronic asthmatics are usually worse in winter months, because of the increased frequency of viral infections.

PREVENTION

Avoidance of allergens

There are few instances, in which a single agent can be identified as the cause for attacks of asthma. These allergens include grass pollens, mites, animal dander, drugs, industrial chemicals such as isocyanates and certain articles of diet. The majority of patients are hypersensitive to a wide range of allergens and attempts to avoid all of them are impracticable.

MATERIALS AND METHODS

Clinical Study

The clinical study of **Swasakasam** was carried out during the year 2007-2008 at the Post Graduate Department of Pothu Maruthuvam, Government Siddha Medical College, Palayamkottai. In this study, twenty patients of both sexes were selected in the Out-Patient Department, admitted in the In-Patient ward and were treated with the trial medicine and guided and clearly observed under the supervision of Professor, Reader and Assistant Lecturer in the Post Graduate Department of Pothu Maruthuvam. After discharge, some patients were followed as the Out-patients in the Out-patients Departments. The medicine was also subjected to trial with twenty Out-patients in the Out-patients department after detailed investigation, under the guidance and observation of Professor, Reader and Assistant Lecturer.

Selection of The Patients

The patients were selected on the basis of the clinical findings of cough with expectoration, dysnea, tightness of chest, wheezing, hardly expectoration of scanty, mucoid sputum.

Detailed history of the patient contains past, personal and family histories, socio-economic status, diet, habits, occupational history, exposure to chemical hazards, smoke, dust and cold.

DIAGNOSIS

Siddha methods of diagnosis were employed with the following methods; Mukkuttra nilaigal, En vagai Thervugal, Nilam, Kaalam, Udalkattugal, Poriyal arithal, Pulanal arithal and Vinathal.

INVESTIGATIONS

The following laboratory investigations were done in the college hospital for all the patients.

1. Blood test (TC, DC, ESR, HB %)
2. Urine Analysis (Albumin, Sugar, Deposits)
3. Motion test (Ova, Cyst)
4. Sputum for AFB
5. X-ray chest (PA View)
6. Mantoux investigation
7. Peak Expiratory Flow Rate.

Special investigation like pulmonary function test (spirometry) was done in the private hospital for some patients because of the limited infra-structures within the college hospital, by the author.

To establish the efficacy of the trial medicine, biochemical analysis and pharmacological studies were conducted in the Department of Biochemical and Pharmacology separately in the Government Siddha Medical College, Palayamkottai and anti-bacterial activity was done at Malar Micro Diagnostic Centre, Palayamkottai.

TREATMENT

The trial medicine used in the present clinical study is **“KASHA CHOORANAM-1gm and ANNA PAVALA CHENDURAM -100mg”** (thrice daily with honey, before meals). All the patients were advised strictly to follow the Patthiyam (Dietary Regimen), Pranayamam and Yogic Exercise was also prescribed for the speedy recovery of “Swasakasam”.

RESULTS AND OBSERVATION

Results were observed with respect to the following criteria

1. Sex Distribution
2. Age Distribution
3. Kaalam Distribution
4. Religion Distribution
5. Thina Distribution
6. Paruva Kaalam Distribution
7. Sirupoludhu Distribution
8. Occupation Distribution
9. Socio - economic status
10. Aetiological factors
11. Mode of Onset
12. Clinical features
13. Duration of Illness
14. Other System involvement
15. Complications in Other System
16. Family history
17. Diet factor
18. Habits
19. Gnanendhiriyam (Imporigal)
20. Kanmendhiriyam
21. Kosam
22. Mukkutram a)Vadham b)Pitham c) Kabam
23. Ezhu Udal Kattugal
24. Envagai Thervugal
25. Neerkuri
26. Neikuri
27. Laboratory Analysis
28. Gradation of Results

For this study 20 In-patients and 20 Out-patients were selected.

1. SEX DISTRIBUTION

Table 1 illustrates the distribution of Sex

Table 1

S. No	Sex	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Male	10	50%	12	60%
2.	Female	10	50%	8	40%

The table showed that males were affected more than females.

2. AGE DISTRIBUTION

Table 2 illustrates the distribution of Age

Table 2

S. No	Age in years	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	0-20	-	-	2	10%
2.	21-30	1	5%	4	20%
3.	31-40	2	10%	2	10%
4.	41-50	4	20%	5	25%
5.	51-60	3	15%	4	20%
6.	61 and above	10	50%	3	15%

The table showed predominance of distribution in the age group 61 and above years among the In – Patients and 41-50 years among the Out- Patients.

3. KAALAM DISTRIBUTION

Table 3 illustrates the distribution of Kaalam

Table - 3

S. No	Kaalam	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vadha Kaalam (1-33 Years)	1	5%	6	30%
2.	Pitha Kaalam (34-66 years)	13	65%	12	60%
3.	Kaba Kaalam (67-100 years)	6	30%	2	10%

The table showed the increased incidence of the disease in the Pitha Kaalam i.e 34 - 66 years.

4. RELIGION DISTRIBUTION

Table 4 illustrates the distribution of Religion among the patients

Table 4

S. No	Religion	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Hindus	19	95%	17	85%
2.	Muslims	-	-	2	10%
3.	Christians	1	5%	1	5%

Among the In-patients 95% were Hindus, 5% were Christians.

Among the Out-patients 85% were Hindus, 10% were Muslims and 5% were Christians.

5. THINAI DISTRIBUTION

Table 5 illustrates the distribution of the disease among Thina

Table 5

S. No	Thinai	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Kurinji	3	15%	1	5%
2.	Mullai	-	-	-	-
3.	Marutham	15	75%	16	80%
4.	Neithal	2	10%	3	15%
5.	Palai	-	-	-	-

The table indicated that Marutham was the place of incidence of the disease.

6. PARUVAKAALAM DISTRIBUTION

Table 6 illustrates the distribution of the disease among the Paruva Kaalam

Table 6

S. No	Paruva Kaalam	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Kaar Kaalam	-	-	-	-
2.	Koothir Kaalam	2	10%	4	20%
3.	Munpani Kaalam	5	25%	11	55%
4.	Pinpani Kaalam	9	45%	4	20%
5.	Elavenil Kaalam	4	20%	1	5%
6.	Muthuvenil Kaalam	-	-	-	-

The table showed the prevalence of disease under pinpani Kaalam among the In-patients and Munpani kaalam among the Out – Patients.

7. SIRUPOLUDHU DISTRIBUTION

Table 7 illustrates the distribution of the disease among the Sirupoludhu.

Table 7

S. No	Sirupoludhu	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vaigarai	17	85%	18	90%
2.	Pagal	-	-	-	-
3.	Nan Pagal	-	-	-	-
4.	Pirpagal	-	-	-	-
5.	Maalai	3	15%	2	10%
6	Yamam	-	-	-	-

The table showed the prevalence of disease during Vaigarai Poludhu.

8. OCCUPATION

Table 8 illustrates the distribution of Occupation among the patients

Table 8

S. No	Occupation	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Agri labour	7	35%	5	25%
2.	Mill Worker	4	20%	4	20%
3.	student	-	-	1	5%
4.	Scavenger	3	15%	1	5%
5.	Auto Driver	-	-	1	5%
6.	Teacher	-	-	2	10%
7.	Painter	1	5%	-	-
8.	House Wives	3	15%	3	15%
9.	Beedi Worker	2	10%	2	10%
10	Nurse	-	-	1	5%

The table indicated increased incidence of the disease in Agricultural labours, House wives and Mill workers.

9. SOCIO - ECONOMIC STATUS

Table 9 illustrates the Socio - Economic Status of the patients

Table 9

S. No	Socio - Economic Status	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Rich	-	-	-	-
2.	Middle Class	3	15%	1	5%
3.	Poor	17	85%	19	95%

85% of In – patients and 95 % Out – patients were economically poor and 15% of In - patients and 5% of the Out - patients were middle class.

10. AETIOLOGICAL FACTOR (ALLERGEN)

Table 10 illustrates the Aetiological Factor for the disease

Table 10

S. No	Aetiology	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Dust	4	20%	6	30%
2.	Smoke	2	10%	2	10%
3.	Husks of grains	3	15%	6	30%
4.	Dust and cold exposure	4	20%	2	10%
5.	Husks of grains and cold exposure	5	25%	-	-
6.	Others	2	10%	4	20%

The above table showed that, dust and cold, dust, husk of grains and cold exposure and husk of grains were the main aetiological factors among these patients.

11. MODE OF ONSET

Table 11 illustrates the Mode of Onset of the disease

Table 11

S. No	Mode of onset	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Sudden	-	-	-	-
2.	Gradual	20	100%	20	100%

The table showed that the mode of onset was gradual in all the 100% of both In-patients and Out- Patients.

12. CLINICAL FEATURES

Table 12 illustrates the distribution of Clinical Features.

Table 12

S. No	Clinical Features	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Running Nose	16	80%	17	85%
2.	Sneeze	15	75%	14	70%
3.	Tightness of Chest	20	100%	20	100%
4.	Wheeze	20	100%	20	100%
5.	Cough with Expectorations	20	100%	20	100%
6.	Fever	4	20%	2	10%
7.	Others				
	(i) Tachycardia	5	25%	4	20%
	(ii) Urticaria	1	5%	1	5%
	(iii) Clubbing	1	5%	1	5%
	(iv) Cyanosis	-	-	-	-

The table shows that most of the patients had cough with expectoration, wheeze,tightness of chest,running nose.

13. DURATION OF ILLNESS

Table 13 illustrates the distribution of Duration of Illness

Table 13

S. No	Duration of Illness	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Below 3 months	4	20%	5	25%
2.	3 - 6 months	4	20%	4	20%
3.	6 months – 1 year	6	30%	5	25%
4.	1 - 2 years	3	15%	2	10%
5.	2 - 3 years	3	15%	4	20%

Among the In - Patients, 20 % each incidence for the duration of below 3 months, and 3 – 6 months, 30% incidence for the duration of 6 months – 1 year, 15% each incidents for the duration of 1-2 years and 2 - 3 years.

Among the Out - Patients, 25% each incidence for the duration of below 3 months, and 6months – 1 year, 20% each for the duration of 3 - 6 months and 2 - 3 years, 10% incidence for the duration of 1-2 years.

14. OTHER SYSTEM INVOLVEMENT

Table 14 illustrates the distribution of co-existing symptoms involving others system.

Table 14

S. No	System	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Cardio Vascular System	-	-	-	-
2.	Gastro Intestinal System	6	30%	4	20%
3.	Musculo Skeletal System	10	50%	4	20%
4.	Central Nervous System	-	-	-	-

The table illustrated that musculo skeletal system, gastro intestinal system was affected more than any other system with this disease.

15. COMPLICATIONS IN OTHER SYSTEM

Table 15 illustrates the distribution of complications involving others system.

Table - 15

S. No	System	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Cardio Vascular System	-	-	-	-
2.	Gastro Intestinal System	-	-	-	-
3.	Musculo Skeletal System	-	-	-	-
4.	Central Nervous System	-	-	-	-

All the In-patients and Out-patients had no complications involving other systems.

16. FAMILY HISTORY

Table16 illustrates the distribution of Family History

Table 16

S. No	Family History	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Positive	5	25%	6	30%
2.	Negative	15	75%	14	70%

The table showed that most of the patients had negative family history.

17. DIET

Table 17 illustrates the distribution of diet among the patients

Table 17

S. No	Diet	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vegetarian	2	10%	1	5%
2.	Mixed diet	18	90%	19	95%

The table showed the highest incidence of the disease for the patients with mixed diet.

18. HABITS

Table 18 illustrates the distribution of habits

Table 18

S. No	Habits	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Smoker	8	40%	7	35%
2.	Tobacco Chewer	2	10%	1	5%
3.	Betel nut chewer	2	10%	3	15%
4.	Alcoholic	5	25%	6	30%
5.	No such habits	2	10%	2	10%
6.	Snuff	1	5%	1	5%

The table showed the highest incidence of the disease in the smokers, Alcoholic.

19. IMPORIGAL (GNANENDHIRIYAM)

Table 19 illustrates the distribution of disease with Imporigal.

Table 19

S. No	Imporigal	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Mei	1	5%	1	5%
2.	Vai	-	-	-	-
3.	Kann	8	40%	5	25%
4.	Mookku	16	80%	17	85%
5.	Sevi	1	5%	-	-

The table showed that Mookku was affected in most of the patients.

20. KANMENDHIRIYAM

Table 20 illustrates the distribution of disease with Kanmendhiriyam.

Table 20

S. No	Kanmendhiriyam	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Kai	-	-	-	-
2.	Kaal	8	40%	5	25%
3.	Vai	-	-	-	-
4.	Eruvai	4	20%	6	30%
5.	Karuvai	-	-	-	-

The table showed that Eruvai and Kaal were affected in most of the cases.

21. KOSAM

Table 21 illustrates the distribution of Kosam.

Table 21

S. No	Kosam	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Annamayakosam	6	30%	4	20%
2.	Pranamayakosam	20	100%	20	100%
3.	Manomayakosam	-	-	-	-
4.	Vingyanaamayakosam	-	-	-	-
5.	Anandhamayakosam	-	-	-	-

In all the In-patients and Out- Patients Pranamayakosam were affected. Annamayakosam was affected in 30% of the In-patients as well as 20% of the Out-patients.

22. MUKKUTRAM a.VADHAM b.PITHAM c.KABAM

22.a. VADHAM

Table 22.a illustrates the distribution of Vadha in the disease.

Table 22 .a

S. No	Types of Vadham	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Pranan	20	100%	20	100%
2.	Abanan	4	20%	6	30%
3.	Viyanan	8	40%	5	25%
4.	Udhanan	20	100%	20	100%
5.	Samanan	20	100%	20	100%
6.	Nagan	-	-	-	-
7.	Koorman	8	40%	5	25%
8.	Kirugaran	16	80%	17	85%
9.	Devathathan	9	45%	4	20%
10.	Dhananjeyan	-	-	-	-

The table showed that the Pranan, Udhanan, Samanan were affected completely in this disease.

22.b. PITHAM

Table 22.b illustrates the distribution of Pitha in the disease.

Table 22.b

S. No	Types of Pitham	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Analpitham	4	20%	9	45%
2.	Ranjagapitham	2	10%	1	5%
3.	Sadhagapitham	20	100%	20	100%
4.	Aalosagapitham	8	40%	5	25%
5.	Prasagapitham	1	5%	1	5%

The table showed that the Sadhagapitham was affected in all the patients in this disease.

22. c. KABAM

Table 22.C illustrates the distribution of Kabam in the disease.

Table 22.c

S. No	Types of Kabam	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Avalambagam	20	100%	20	100%
2.	Kilethagam	4	20%	9	45%
3.	Pothagam	-	-	-	-
4.	Tharpagam	-	-	-	-
5.	Sandhigam	8	40%	5	25%

The table showed that the Avalambagam was affected in all the patients in this disease.

23. EZHU UDAL KATTUGAL

Table 23 illustrates the distribution of derangement of Udal Kattugal in the disease.

Table 23

S. No	Ezhu Udal Kattugal	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Saaram	20	100%	20	100%
2.	Senneer	16	80%	13	65%
3.	Oon	-	-	-	-
4.	Enbu	8	40%	5	25%
5.	Kozhuppu	8	40%	5	25%
6.	Moolai	-	-	-	-
7.	Sukkilam / Suronitham	-	-	-	-

The table showed that Saaram and seneer was affected in most of the patients in this disease.

24. EN VAGAI THERVUGAL

Table 24 illustrates the distribution of En Vagai Thervugal in the disease.

Table 24

S. No	En Vagai Thervugal	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Naa	5	25%	7	35%
2.	Niram	6	30%	3	15%
3.	Mozhi	20	100%	20	100%
4.	Vizhi	8	40%	5	25%
5.	Malam	4	20%	6	30%
6.	Moothiram	-	-	-	-
7.	Sparisam	12	60%	10	50%
8.	Naadi				
	a.Vadha Kaba	16	80%	15	75%
	b.Pitha Kaba	1	5%	3	15%
	c.Kaba Vadha	3	15%	2	10%

The table showed that **Mozhi was affected in all the patients in this disease.** Naa, Niram, Malam,Sparisam,Vizhi were affected in most of the patients. In Naadi, Vadha Kaba Naadi showed higher frequency than the others.

25. NEER KURI

Table 25 illustrates the distribution of Neer Kuri in the disease.

Table 25

S. No	Neerkuri	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Niram	1	5%	2	10%
2.	Edai	-	-	-	-
3.	Manam	-	-	-	-
4.	Nurai	-	-	-	-
5.	Enjal	-	-	-	-

Niram was affected in 5% of the In – Patients and 10% of the Out-patients. Edai, Manam, Nurai, Enjal were not affected.

26. NEIKURI

Table 26 illustrates the distribution of Neikuri in the disease.

Table 26

S. No	Neikuri	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vadha Neer	2	10%	4	20%
2.	Pitha Neer	2	10%	1	5%
3.	Kaba Neer	16	80%	15	75%

The table showed that Kaba Neer was found in most of the cases.

27. LABORATORY INVESTIGATIONS

Table 27 – A, 27 – B, 27 – C and 27 – D illustarte the laboratory investigations.

Sputum for AFB and Mantoux test were found to be negative in all the 100% of In-patients and Out-patients.

28. GRADATION OF RESULTS

Table 28 illustrates the Gradation of Results

Table 28

S. No	Result	In-patients		Out-patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Good	14	70%	14	70%
2.	Fair	4	20%	4	20%
3.	Poor	2	10%	2	10%

Good results were found in 70% of the In-patients and in 70% of the Out-patients. Fair results were found in 20% of the In-patients and in 20% of the Out - Patients. Poor results were found in 10% of the In-patients and in 10% of the Out – Patients.

DISCUSSION

Millions of people are affected all over the world by the disease Swasakasam, which is similar to Bronchial Asthma, occurring due to pollution of environment and changes in life style.

Efficacy of siddha system in curing the respiratory disease prompted the author to carry out clinical and scientific study in this subject.

For the clinical study 20 selected patients were admitted as In-patients in Post Graduate Department of Pothu Marthuvam and were treated with the trial medicine. After discharge all the twenty patients were followed as the Out-patients.

The medicine was also trialed with 20 Out-patients in the Out-patients Department of Pothu Maruthuvam.

The results were clearly observed and recorded under the supervision of Professor, Reader and Assistant lecturer. The observed results were discussed here.

1. Sex Distribution

Among the In-patients 50% each of the males and females were affected. Among the Out-patients 60% of the males were affected and 40% of the females were affected.

This indicated that males were mostly affected by the disease than females and this may be due to increased exposure of males to the polluted environment .

2. Age Distribution

Among the In-patients 50% of the patients were affected in the age groups of 61 and above years, 20% of the patients were affected in the

age group of 41-50 years, 15% of the patients were affected in the age group of 51-60 years, 10% of the patients were affected in the age group of 31-40 years, 5% of the patients were affected in the age group of 21-30 years.

Among the Out-patients 25% of the patients were affected in the age group of 41-50 years and 20% of each of the patients were affected in the age group of 51-60 years and 21-30 years, 15% of the patients were affected in the age group of 61 and above years and 10% each of the patients were affected in the age group of 31-40 years and 0-20 years.

It showed that increased incidence of the patients who came for treatment fall under the age group of 61 and above in the In-patients and 41-50 years among the out patients.

It indicated that the increased incidence of the disease during the old age was due to decreasing lung capacity, lung function and immunity.

3. Kaalam Distribution

Among the In - Patients 65% were affected in Pitha Kaalam, 30% were affected in Kaba Kaalam and 5% were affected in Vadha Kaalam.

Among the Out - Patients 60% were affected in Pitha Kaalam, 10% were affected in Kaba Kaalam, and 30% were affected in Vadha Kaalam.

The table showed the increased incidence of the disease in the Pitha Kaalam i.e 34 - 66 years.

4. Religion Distribution

Among the In-patients 95% were Hindus, 5% were Christians.

Among the Out-patients 85% were Hindus, 10% were Muslims and 5% were Christians.

5. Thinai Distribution

Among the In-patients 75% belonged to the Marutham (i.e. plain & its surroundings) and 15% belonged to the Kurinji (i.e. mountains & its surroundings) and 10% belonged to the Neithal (i.e. sea & its surroundings).

Among the Out-patients, 80% belonged to the Marutham (i.e. plain & its surroundings) and 15% belonged to the Neithal (i.e. sea & its surroundings) and 5% belongs to the Kurinji (i.e. mountain & its surroundings)

According to the literature Marutha Nilam is free from disease.

According to Noiilla Neri text.

“kUjepy ed;dPh; tsnkhd;iwf; nfhz;NI
nghUjepy khjpa Neha; Nghf;Fk;”
“ePhpd; Fznky;yhk; kz;zpd; Fzky;yhy;
kw;W Kz;NI”

The utilization of land is due to its water source.

நீரின் வழியாக மட்டுமின்றி வெப்பம், காற்று இவைகளின்
வழியாகவும் நிலத்தின் பயனை அடையலாம்.

The utilization of land is along with water, heat and air.

As these three were affected in these days due to pollution, the disease occurs in this area also.

The observation indicated that the incidence of the disease is highest in Marutha Nilam. (i.e. plain & its surroundings).

6. Paruva Kaalam Distribution

Among the In-patients 45% of the incidence of the disease fall under the Pinpani Kaalam i.e. Maasi & Panguni (February & March) and 25% of the incidence fall under the Munpani Kaalam i.e. Margali & Thai (December & January) and 20% of the incidence fall under the Elavenic Kaalam i.e. Chithirai

& Vaigasi (April & May) and 10% of the incidence fall under the Koothir Kaalam i.e. Iypasi & Karthigai (October & November).

Among the Out-patients 55% of the incidence fall under the Munpani Kaalam i.e. Margali & Thai (December & January) and 20% each of the incidence fall under the Pinpani Kaalam i.e. Maasi & Panguni (February & March) and Koothir Kaalam i.e. Iypasi & Karthigai (October & November) and 5% of the incidence fall under the Elavenil Kaalam i.e. Chithirai & Vaigasi (April & May).

7. Sirupoludhu Distribution

Among the In-patients 85% of the incidence of the disease occurs during vaigarai poludhu i.e. 4 hours before sunrise and 15% of the incidence of the disease occurs during Maalai Poludhu i.e. 4 hours of the sunset.

Among the Out-patients 90% of the incidence of the disease occurs during vaigarai poludhu i.e. 4 hours before the sunrise and 10% of the incidence of the disease occurs during Maalai Poludhu i.e. 4 hours of the sunset.

It was observed that the incidence of this disease occur during early morning.

8. Occupational Distribution

Among the In-patients 35% belongs to Agricultural labours, 20% were Mill workers, 15% each were House wives and scavenger, 10% were Beedi workers and 5% of were painter.

Among the Out-patients 25% were Agricultural labours, 20% were Mill workers , 10 % each were beedi worker and techer, 15 % were house wives and 5% each were scavenger, auto driver, student ,nurse.This observation indicated increased incidence of the disease in Agricultural labours, House wives and Mill workers.

9. Distribution of Socio-Economic status

Among the In-patients 85% belonged to the poor Socio-Economic status and 15% belonged to middle class.

Among the Out-patients 95% belonged to the poor socio economic status and 5% belonged to middle class.

This observation indicated the increased incidence of the disease in poor socio-economic status.

10. Distribution of Aetiological factor

Among the In-patients, 20% each of the patients had dust, dust and cold exposure collectively as their aetiological factor, 25% of the patients had husks of grains and cold exposure collectively as their aetiological factors, 15% had husks of grains collectively as their aetiological factor and 10% had smoke collectively as their aetiological factor. Another 10% of the patients had other factors such as cotton fibre and exposure to sulphur and lead as their aetiological factors.

Among the Out-patients, 30% each had dust and husks of grains collectively as their aetiological factor, 10% each had smoke, dust and cold exposure collectively as their aetiological factor. Another 20% had other factors such as cotton fibre collectively as their aetiological factors.

Above data illustrated dust and cold exposure, husks of grains and dust were the aetiological factors among the patients.

According to literature, the aetiological factors are excessive inhalation of smoke, cold climate, noisy wind, husks of grains, inhalation of irritant fragrance.

Thus the above data coincided with the literature.

11. Distribution of Mode of Onset

The observation illustrated the mode of onset in all the 100% of the In-patients and Out-patients were gradual.

12. Distribution of Clinical Features

The data from the observation showed that 100% of incidence of tightness of chest, wheeze, cough with scanty expectoration, in both In-patients and Out-patients.

Running nose was present in 80% of the In-patients and 85% of the Out-patients. Sneezes were present in 75% of the In-patients and 70% of the Out-patients. Tachycardia was present in 25% of the In-patients and 20% of the Out-patients during the time of episode. Fever was present in 20% of the In-patients and 10% of the Out-patients, urticaria was present in 5% of the In-patients and Out-patients, clubbing was present in 5% of the In-patients and Out-patients.

13. Distribution of Duration of illness

Among the In - Patients, 20 % each incidence for the duration of below 3 months, and 3 – 6 months, 30% incidence for the duration of 6 months – 1 year, 15% each incidents for the duration of 1-2 years and 2 - 3 years.

Among the Out - Patients, 25% each incidence for the duration of below 3-months, and 6months – 1 year, 20% each for the duration of 3 - 6 months and 2 - 3 years, 10% incidence for the duration of 1-2 years.

The data illustrated the highest incidence of duration of illness among, both the In-patients and the Out-patients were below 3 months and 6 months – 1 year.

14. Involvement of Other Systems

Among the In-patients 30% of the patients had the involvement of Gastrointestinal system and 50% of the patients had the involvement of Musculoskeletal system.

Among the Out-patients 20% each of the patients had the involvement of Gastrointestinal system and the involvement of Musculoskeletal system each.

In both In-patients, Out-patients musculo skeletal system was affected more than any other system with the disease.

The symptoms that noticed among the patients were mainly joint pain may be due to aging.

15. Complication in Other System

All the In-patients and Out-patients had no complication involving other systems.

16. Family History

Among the In-patients, 75% of the patients had negative family history and 25% of the patients had positive family history.

Among the Out-patients, 70% of the patients had negative family history and 30% of the patients had positive family history.

It is showed that most of the patients had negative family history.

17. Diet

The observations illustrated that among the In-patients 90% of them had mixed diet and 10% had vegetarian diet.

Among the Out-patients, 95% of the patients had mixed diet and 5% had vegetarian diet.

It indicated that the disease was predominant in the mixed diet habitants.

According Yugi Vaidhya Chinthamani, the dietary factors that cause the disease are taking non-vegetarian diet and taking improperly cooked food.

Here the observations coincide with Yugi's concept.

18. Habits

Among the In-patients 40% of the patients were smokers, 25% were alcoholics, 10% each were tobacco chewers, betel nut chewers, 5% were snuff. Other 10% had no such habits.

Among the Out-patients 35% were smokers 30% were alcoholics, 15% were betel nut chewers and 5% each were tobacco chewers and snuff. Another 10% had no such habits.

The disease was predominant in smokers and Alcoholic.

19. Imporigal

Among the In-patients mooku (Nose) was affected in 80% of the In-patients i.e. anosmia due to running nose. Kann was affected in 40% of the patients. Kann (Eye) had dull vision may be due to aging and was red due to conjunctivitis. Mei was affected in 5% of the patients. Mei (Skin) had itching all over the body during episodes. Sevi (ear) was affected in 5% of the patients. Sevi had hearing impaired due to aging.

Among the out- patients, mooku was affected in 85% the Out-patients Kann was affected in 25% of the patients and mei was affected in 5% of the patients. Vai and Sevi were not affected.

20. Kanmendhiriyam

It was illustrated that among the In-patients, Kaal was affected in 40% of the patients. They had knee joint pain may be due to aging. Eruvai was affected in 20% of the patients. They had constipation. Kai, vai, and karuvai were not affected.

Among the Out-patients, kaal was affected in 25% of the patients & Eruvai was affected in 30% of the patients. kai, vai and Karuvai were not affected.

This showed that Eruvai and kaal were affected in most of the cases.

21. Kosam

It was illustrated that Pranamaya kosam was affected in all the patients of both the In-patients and Out -patients. pranamayakosam were affected due to cough with expectoration, tightness of chest, wheeze.

It was illustrated that Annamaya kosam was affected in 30% of the In-patients and 20% of the Out-patients. Annamaya kosam were affected due to indigestion.

22. Mukkutram a. Vadham b.Pitham c. Kabam

a. Vadham

Pranan, udhanan, samanana were affected in all the 100% of the In-patients and Out-patients. Abanan was affected in 20% of the In-patients and 30% of the Out-patients. Viyanan was affected in 40% of the In-patients, and 25% of the Out-patients. Koorman was affected in 40% of the In-patients, and 25% of the Out-patients. Kirugaren was affected in 80% of the In-patients, and 85% of the Out-patients. Devathathan was affected in 45% of the In-patients and 20% of the Out-patients.

Pranan is responsible for respiration. In swasakasam, this vayu was affected leading to difficulty in breathing, cough, wheeze is also caused by pranan.

Viyanan main function is movements of the joints. In clinical trial, this vayu was affected leading to knee joint pain which may be due to aging.

Udhanan is responsible for speech, strength of the mind and the body . Since there was low pitched voice, decreased in strength of body and mind . This vayu was affected in this disease.

Samanan is responsible for controlling other vayus and digestion. Since samanana cannot control other vayus, it affected in this disease,

Koorman is responsible for vision. In the clinical trial this vayu was affected due to aging.

Kirugaran is responsible for appetite, sneeze, cough and running nose. It was affected in this disease.

Devathathan is responsible for tiredness after sleep and emotion. This was due to severity of the asthmatic episode.

b. Pitham

Sadhaga pitham was affected in all the 100% of both In-patients and Out-patients. Anal pitham was affected in 20% of the In-patients and 45% of the Out-patients. Aalosaga pitham was affected in 40% of the In-patients and 25% of the Out-patients. Prasaga pitham was affected in 5% of the In-patients and 5% of the Out-patients. Ranjaga pitham was affected in 10% of the In-patients and 5% of the Out-patients.

Sadhaga pitham influences the normal day to day activities with the help of mind and brain, since dyspnoea and restlessness is present, this pitha was affected in this disease.

Anal pitham is responsible for appetite, Since there was loss of appetite. In the clinical trial the pitha was affected.

Ranjaga pitham is responsible for coloring of the blood. It may be affected if the patient has lassitude, pallor, weakness, anorexia, indigestion..

Aalosaga pitham is responsible for clear vision. Since, there was diminished vision, this was affected. It may be due to their aging.

Prasaga pitham is responsible for skin complexion, since there was itching in any part of the body, this was affected.

c. Kabam

Avalambagam was affected in all the 100% of both In-patients and Out-patients. Sandhigam was affected in 40% of the In-patients and 25% of the Out-patients. Kilethagam was affected in 20% of the In-patients and 45% of the Out-patients.

Avalambagam is residing in lungs and helps other four types of kaba to function. It was deranged due to the presence of tightness of chest, cough, wheezing and dyspnoea.

Kilethagam is present in the stomach and gives moisture to the food materials and also helps in digestion. It was deranged due to indigestion.

Sandhigam resides in the joints and helps for movement. Since, there was Joint pain, it was affected. It was may be due to their aging.

23. Ezhu udal kattugal

In Ezhu udal kattugal, saaram was affected in all the 100% of the In-patients and Out-patients. Enbu and Kozhuppu were affected in 40% of the In-patients and 25% of the out- patients. Senner was affected in 80% of the In-patients and 65% of the Out-patients.

Saaram strengthens the body and mind, Since, there is loss of appetite and strengthless causing body tiredness due to wheeze, cough, tightness of chest.

Enbu and Kozhuppu are responsible for the movements of the body and gives lubrication to the joint cavities. Since, there was joint pain, these two were affected may be due to aging.

24. En vagai thervugal

Mozhi was affected in all the 100% of both In-patients and Out-patients due to low pitched voice and difficulty in speech. Naa was affected in 25% of

the In-patients and 35% of the Out-patients due to coated tongue, pallor tongue. Vizhi was affected in 40% of the In-patients and 25% of the Out-patients due to diminished vision. Malam was affected in 20% of the In-patients and 30% of the out- patients due to constipation. Niram was affected in 30% of the In-patients and 15% of the Out-patients due to pallor. Sparisam was affected in 60% of the In-patients and 50% of the Out-patients due to sweating.

In Naadi, 80% of the In-patients and 75% of the Out-patients had vadha kaba naadi. 5% of the In-patients and 15% of the out- patients had pitha kaba naadi. 15% of the In-patients and 10% of the Out-patients had kaba vadha naadi.

25. Neerkuri

Niram was affected in 5% of the In-patients and 10% of the Out-patients due to vitiated kaba. Edai, Manam, Nurai and Enjal were not affected.

26. Neikuri

In Neikuri 10% of the In-patients and 20% of the out- patients had vadha neer. 10% of the In-patients and 5% of the out –patients had pitha neer. 80% of the In-patients and 75% of the Out-patients had kaba neer.

This showed the kaba neer was found in most of the cases.

27. Laboratory Investigations

Routine investigations of blood and urine were done during the admission and at the end of the treatment for every case.

Blood, Urea and Serum cholesterol were found to be in normal range before and after treatment.

X-ray chest PA view showed normal for 95% of the cases and 5% of the cases showed bronchitis in In-patients.

X-ray chest PA view showed normal for 90% of the cases and 10% of the cases showed bronchitis in Out-patients.

The peak flow meter reading showed diurnal variation. The peak flow meter ranged from 70 l/min to 200 l/min before treatment among the In-patients. After treatment it ranged from 150 l/min to 380 l/min.

Among the Out-patients the peak flow meter reading ranged from 70 l/min to 180 l/min before treatment and after treatment it ranged from 150 l/min to 380 l/min.

Pulmonary Function Tests are done for three patients two from In-patients and one from Out-patients. The results showed clinically co-related obstructive lung disease.

Urine examination in In-patients and Out-patients showed nil albumin. But 5% of the In-patients showed ++ in the urine sugar and after treatment it was nil. 10% of the In-patients showed pus cells and epithelial cells in their urine and after treatment it was NAD (Nothing Abnormal detectable) with appropriate medicine.

Urine examination in Out-patients showed 10% of the patients had epithelial cells, 5% had pus cells and after treatment it was NAD, with appropriate medicine.

Blood investigations of In-patients showed total count of WBC with in the normal range. Eosinophils count was increased and ranged from 3-12% cells before treatment and after treatment it ranged 1% to 5%. ESR (Erythrocyte Sedimentation Rate) was raised in before treatment, and the after treatment was reduced. 10% of In-patients Hemoglobin content was decreased in before treatment and the after treatment was normal.

Blood investigation of Out-patients showed TC with in the normal range. Eosinophils count was raised and should the range of 3% to 14% cells before treatment, and after treatment it ranged 1% to 8%.

Before treatment Hemoglobin content was decreased in 5% of the Out-patients and the after treatment was normal

Motion test revealed that 100% of both In-patients and Out-patients had nil results before treatment as well as after treatment.

Sputum examination was found to be negative for all the 100% of both the In-patients and Out-patients.

Mantoux test was found to be negative for all the 100% of both the In-patients and Out-patients.

Modern Medicine Comparison

According to modern medicine, the aetiological factors for the disease are exposure to dust, smoke, pollens, grains, chemicals and cold exposure, stress and food habits.

In our literature, Yugi said more or less the same reasons for the disease.

The signs and symptoms of the disease Bronchial Asthma are closely matched with Swasakasam as explained by yugi muni.

Treatment

On the previous day of treatment, laxative Nilvagai chooranam 5gm with hot water at the bed time after starting the internal medicine.

On the first day, the trial medicine “Kasha Chooranam – 1gm and Annapavala Chenduram – 100mg thrice daily with honey before meals was prescribed and was given till the end of their treatment.

Diet regimen

Patients were advised to avoid watery vegetables and fruits, bitter guard, jack fruit, dry fish, fish, onion, butter, curd, coconut, cold water and cold food.

Patients were recommended to take vegetables such as brinjal drumstick, ginger, garlic, raddish, greens, such as Eclipta alba etc., and goat ghee.

PRANAYAMAM

Patients were advised to do Pranayamam breathing exercise 20 counts twice daily for better results.

YOGA THERPY

Yogasanas such as Bujangasanam, Chakrasanam, Machasanam, Mayurasanam, PathaHasthasanam, Arai Machayenrhirasanam, Trikonasanam and Savasanam were advised to be practiced for quick relief.

Gradation of results

Good results showed that the asthmatic episodes in a week are reduced more than 90% and markedly increased Peak Flow Meter Rate (PFMR), after treatment 70% each of the In-patients and Out-patients were observed good results.

Fair results showed that the asthmatic episodes in a week are reduced more than 50% and greatly increased Peak Flow Meter Rate (PFMR), after treatment 20% of each the In-patients and Out-patients were observed fair results.

Poor results showed that the asthmatic episodes in a week are reduced below 40% or no improvement and only a minimal increase in PFMR after treatment 10% each of the In-patients and Out-patients were observed in poor results.

Biochemical analysis for kasha Chooranam showed the presence of Ferrous iron, Starch, unsaturated compound, Aminoacid.

Biochemical analysis for Anna pavala chenduram showed the presence of calcium, Sulphate, Chloride, Ferric iron, Ferrous iron.

KASHA CHOORANAM AND ANNA PAVALA CHENDURAM

Pharmacological analysis revealed that the trial medicine **Kasha chooranam** and **Anna pavala Chenduram** had significant Anti-histamine Anti-spasmodic and Anti-inflammation activities.

Anti – microbial study showed the trial medicine is sensitized to **staphylococcus aureus** and **pseudomonas aeragonosa**. Clinically no side effects and adverse effects were noted for the maximum 60 days of therapy.

SUMMARY

Swasakasam is the common respiratory disease seen in day to day clinical practice.

Sincerity, charity and skill are the basis of practice of medicine. Further loving tender care is essential for winning co-operation and confidence of the patients for the ultimate recovery.

The trial medicine for Kasha Chooranam the ingredients of this medicine has a potent anti kaba action. Also the ingredients of Annapavala Chenduram namely Annapethi and Kodi pavalam has been recommended as a remedy for swasakasam in various siddha literature.

The aetiology, pathology, classification, clinical features, diagnosis, complications, prognosis, treatment and preventive measures were selected from siddha and modern systems of medicine.

In this study, 20 patients of both sexes of varying age groups were selected as In-patients and 20 patients as Out-patients.

From the observation and results, we were clear that the disease was common in the following aspects.

- ❖ Males were mostly affected than females. Age incidence has commonly found in all decades. Increased during the occupational period.
- ❖ Most of the patients were poor socio-economic status.
- ❖ Hindus were mostly affected.
- ❖ Majority of the cases were affected in Munpani Kaalam (Margali & Thai) and Pinpani Kaalam (Maasi & Panguni)

- ❖ Most of the cases got the acute symptoms during Vaigarai poludhu i.e., 4 hours before sunrise. Most of the patients belonged to the Thina Marutham.
- ❖ In the occupation, agricultural labours, mill workers and house wives were mostly affected.
- ❖ Aetiological factors were mostly dust and cold exposure, husks of grains and cold exposure, dust, husks of grains.
- ❖ All the patients had gradual onset of the disease.
- ❖ All the patients were affected with clinical features of cough with scanty mucoid sputum, tightness of chest, Wheeze.
- ❖ Duration of illness ranged up to 3 months.
- ❖ Musculo skeletal system and Gastro intestinal system was mostly affected.
- ❖ 75% of the In-patients and 70% of the Out-patients had negative family history.
- ❖ Smokers were affected in this disease
- ❖ In imporigal mooku was affected in most of the cases of In-patients and Out-patients.
- ❖ In Kanmendhiriyam, kaal and eruvai was mostly affected.
- ❖ In kosam, Pranamayakosam were affected in all the 100% of both the In-patients and Out-patients, Annamaya kosam were affected in 30% of the In-patients and 20% of the Out-patients.
- ❖ **In Mukkutram**
 - In Vadham – Pranam, Udhanam, Samanam were affected in all the 100% of the both In-patients and Out-patients.

Abanan, Viyanan, Koorman, Kirugaran, Devathathan were affected in many cases of both In-patients and Out-patients.

- In Pitham – Sadhaga Pitham was affected in all the 100% of the cases of both In-patients and Out-patients.

Anal Pitham and Aalosagapitham was affected in most of the cases.

- In Kabam – Avalambagam was affected in all the cases. Sandhigam and Kilethagam was affected in few cases.

❖ In Ezhu Udal kattugal – Saaram was affected in all the 100% of the cases of In-patients and Out-patients.

Senneer, Enbu and Kozhuppu was affected in few cases.

❖ In Envagai thervugal,

- Mozhi was affected in all the 100% of both the In-patients and Out-patients.
- Naa, vizhi and Malam, Niram and Sparisam were affected in most of the patients.
- Neerkuri showed transparent urine.
- Neikuri showed derangement of Mukkutram. Kaba neer was found in most of the cases.

❖ Laboratory investigations showed normal blood urea, serum cholesterol, normal TC count, raised Eosinophil count and ESR. Blood sugar normal in most of the cases.

❖ Urine analysis showed Epithelial cells in few cases and it was nothing abnormal detectable (NAD) after treatment, with appropriate medicine.

❖ Sputum analysis showed negative AFB (Acid Fast Bacilli) and Mantoux test was negative in all the patients.

- ❖ X-ray chest PA view was normal in most of the cases.
- ❖ Peak flow meter reading among the In-patients ranged from 70 l/min to 200 l/min before treatment and 150 l/min to 380l/min after treatment. Among the Out-patients it ranged from 70 l/min to 180 l/min before treatment and after treatment it ranged from 150 l/min to 380 l/min it showed better prognosis of the disease.
- ❖ This efficacy of the trial medicine, **Kasha Chooranam** and **Annapavala Chenduram** was studied and observed during the dissertation period.
- ❖ Pulmonary Function Tests showed the results for three patients they had obstructive lung disease.
- ❖ All the patients were advised to follow strict diet restrictions and advised to practice Pranayama and Yoga therapy for fast relief.
- ❖ Clinically 70% of the patients showed good results.
- ❖ No side effects and adverse effects were noticed during the period of study.
- ❖ Bio-chemical Analysis for **Kasha Chooranam** showed the presence of Ferrous iron, Starch, unsaturated compound and amino acids.
- ❖ Biochemical Analysis for **Annapavala Chenduram** showed the presence of calcium, sulphate, chloride, ferric iron and ferrous iron.
- ❖ Pharmacological analysis showed that the trial medicine had significant anti-histamine, anti-Spasmodic and anti-inflammation activities.
- ❖ **Anti-microbial studies** showed that the drug Kasha chooranam and Anna pavala chenduram was sensitized to the **staphylococcus aureus** and **pseudomonas aeruginosa**.

CONCLUSION

❖ 70% of the patients showed good results, 20% showed fair results and 10% showed poor results in this trial study.

❖ The trial medicine kasha chooranam has the tastes of Kaippu, Karppu and Siru inippu altogether.

Suvai - kaippu, Karppu and Siru inippu

Thanmai - Veppam

Pirivu - Kaarppu

As the tastes kaippu, karppu both antagonise the excessive kaba, and inippu has the smoothening effect on throat which has been irritated by the chronic cough, kasha chooranam act as an Anti-kaba medicine.

Anna pavala chenduram namely Anna pethi and kodi pavalam has been recommended as remedy for swasakasam in various siddha text. Anna pavala chenduram act as an Anti- kaba medicine.

And the trial medicine has its Thanmai as veppam. All the veppam natured drugs will the excessive kaba in kaba patients. This medicine also acts as an Anti-kaba medicine

After ingestion while the trial medicine reaches the gastric juice, it will change into the vibagam- kaarppu. At this stage also, the medicine will acts as Anti-kaba medicine due to the vibagam kaarppu.

❖ Kasha chooranam and Anna pavala chenduram act as an Anti-kaba medicine.

❖ Thus kasha chooranam and Anna pavala chenduram yield good prognosis in swasakasam patients.

❖ Further follow up of these patients showed sense of well being and complete disappearance of symptoms.

❖ So swasakasam is controllable with **Kasha Chooranam** and **Annapavala Chenduram**.

ANNEXURE - I

PREPARATION OF THE TRIAL MEDICINE

காசச் சூரணம்

சுத்தித்த கண்டுபாரங்கி	-	1 பங்கு
சுத்தித்த சுக்கு	-	1 பங்கு
சுத்தித்த திப்பிலி	-	1 பங்கு

சுத்தி முறைகள்

கண்டுபாரங்கி

இதன் வேரின் மேல் தோலை சீவி எடுத்து பாலில் வேகவைத்து எடுத்து உலர்த்த சுத்தியாகும்.

சுக்கு

இதன் மேல்தோலை சீவி எடுக்க சுத்தியாகும்.

- அனுபவ வைத்திய தேவ ரகசியம் பக்கம் -38.

திப்பிலி

பழரசத்தில் ஊறவைத்து எடுக்க சுத்தியாகும்.

- கோஷாயி அனுபோக வைத்திய பிரம்ம ரகசியம் பக்கம் -9

செய்முறை

சுத்தி செய்த சுக்கு, திப்பிலி, கண்டுபாரங்கி மூன்றையும் இடித்து வெண் துகிலில் வஸ்திரகாயம் செய்து எடுத்து பத்திரப்படுத்த வேண்டும்.

அளவு

1 கிராம்-மூன்று வேளை தினமும்

அனுபானம்

தேன்

தீரும் நோய்கள்

காச சுவாசங்கள் தீரும்

ஆதாரநூல்

- அனுபவ வைத்திய தேவ ரகசியம் பக்கம்-481.

அன்னபவழச்செந்தூரம்

சுத்தித்த அன்னபேதி	-	5 பங்கு
சுத்தித்த கொடி பவழம்	-	1 பங்கு
எலுமிச்சம் பழச்சாறு	-	தேவையான அளவு

சுத்திமுறைகள்

அன்னபேதி

பழச்சாற்றில் ஒரு நாள் முழுவதும் ஊற வைத்து சாறு வற்றுகிற வரையிலும் வைத்தால் சுத்தியாகும்.

- கோஷாயி அனுபோக வைத்திய பிரம்மரகசியம்

பாகம் II பக்கம் -184

கொடி பவழம்

பழச்சாற்றில் ஊற வைத்து கழுவி உலர்த்த சுத்தியாகும்.

- அனுபவ வைத்திய தேவரகசியம் - பக்கம்- 36

செய்முறை

சுத்தித்த அன்னபேதி, கொடி பவழத்தை கல்வத்திலிட்டு பொடித்து எலுமிச்சம் பழச் சாற்றை சிறுக சிறுக வார்த்து இரண்டு சாம நேரம் அரைத்து வில்லை செய்து உலர்த்தி ஒட்டிலிட்டு மேலோடு மூடி 5 சீலைமண் செய்து கவசத்தின் 6 பங்கு எடை வரட்டியிற் புடமிட்டு ஆறின பின்னெடுத்து கல்வத்திலிட்டு பொடித்து முன் போலவே புடமிடவும். இவ்விதமே மூன்றாந் தடவையும் புடமிட்டெடுத்துக் கொள்ள சிறப்பான செந்தூரமாகும்.

அளவு

100-200 மி.கி

துணை மருந்து

தேன்

தீரும் நோய்கள்

இரைப்பிருமல், சயம், இருமல், இரத்த காசம், இரத்த சூடு, பித்த சூடு, மேக சூடு, என்பு சூடு தீரும்.

ஆதாரநூல்

- அனுபோக வைத்திய நவநீதம். பாகம் -III பக்கம் -104

PROPERTIES OF THE TRIAL MEDICINE

கண்டுபாரங்கி

Vernacular Names

Tamil	: Sirutekku, Kandubaranki, Angaravalli, cherutekku.
English	: Beetle killer.
Malayalam	: Cherutekka
Hindi	: Bharangi
Bengali	: Bamanhati, Barnanbati.
Sanskrit	: Barbara, Bharngi
Kannadam	: Bharangi
Telugu	: Gantu Bharag
Common name	: Kandubaranki
Synonyms	: Sirutekku.
Botanical Name	: Clerotendrum serratum
Family	: Verbenaceae.
Parts used	: Root, leaves.

Characters

Suvai	: Kaippu, thuvarppu
Thanmai	: veppam
Pirivu	: Kaarppu
Chemical Constituents	: Saponin, Mannitol & stigmasterd
Therapeutic action	: Stimulant, sedative.

Uses

“கண்டுபா ரங்கியெனுஞ் சிறுதேக குண்டேல்
காலெங்கெ பித்தமெங்கே கபந்தா னெங்கே
தொண்டுதொட்டுத் தொடர்சுவாச காச மெங்கே
சுரமெங்கெ வெறியெங்கே தொணிநோ யெங்கே

மிண்டுபுரி பீந்சநீர்க் கோவை யெங்கே
வெளிநீருண் ணீரெங்கே விறற்கா லெங்கே
அண்டுபடாச் சீதசுர்ங் கடுப்பு மெங்கே
யழலையக நொயெங்கே யறைகு வீரே”.

- குணபாடம்-முதல்பாகம் பொருட் பண்பு நூல்.

பக்கம்-216

It is given in Asthma, Fever, sinusitis and chronic arthritis.

Saraga Samhitha Part II says,

It is used in Asthma, sinusitis, ulcer, cough, fever, oedema and hemorrhagic disorders.

Properties and uses

The kandubaranki root are bitter, Anti-inflammatory, carminative, expectorant, antispasmodic, stimulant.

It is used in cough, Asthma, bronchitis, flatulence, colic and skin disease.

The root is used for Scorpion sting.

-Indian medicinal plant- vol II page -120

The root is used for asthma, bronchitis and other catarrhal affections of the lungs.

Indian material medica-vol I

A.K.Nadkarni.

சுக்கு

Vernacular Names

Tamil	: Shukhu, Chukku
English	: Dry ginger
Telegu	: Sonti
Malayalam	: Chukka, vona-shunti
Punjab	: Sonth
Sanskrit	: Nagaram, visoushada
Kannadam	: ona shunti, shunti
Common Name	: Chukku.
Synonyms	: Vida modeya amirtham, Sundi, sowbannem.
Botanical name	: Zingiber officinale.
Family	: Zingiberaceae
Parts used	: Dried Rhizome.

Characters

Suvai	: Kaarppu
Thanmai	: Veppam
Pirivu	: Kaarppu

Chemical Constituents

Aromatic volatile oil, Zingiberine, gingerol, cineol, camphene, resine, starch,

Therapeutic Action

Stimulant, stomachic, carminative, sialoguague, Aromatic.

Uses

“சுக்குறுஞ் சிறப்பைக் கூறின் சுவைகடு லகுவே ஸ்ருக்த
மிக்குறு முஷ்ணம் வீரியம் வியன்பாக மதுர மாகுஞ்
தக்கிலாக் கபமே வீக்கந் தவிர்ந்திடும் பின்னும் பித்தம்
புக்குழல் சுவாசங் காசம் புரியும்ஸ்லீ பதத்தினோடு”

- பதார்த்த பஞ்ச குண மஞ்சரி. பக்கம்-143.

It is useful in Asthma, cough, swelling,

Properties and uses

The dry ginger is acrid, digestive, stimulant, expectorant, carminative, emolient.

It is useful is asthma, cough, flatulence, diarrhoea, nausea, Vomiting

- Indian medicinal pant-part II

திப்பிலி

Vernacular Names

Tamil	: Tippali, pipili, Tippilli
Malayalam	: Tippli,
Sanskrit	: Pippali, maghadhi, Trikana
Bengal	: Pipli, pepal
Gujarat	: Pipara, pipli, pipal,
Punjab	: Pipal
Malaysia	: Lada, Mula-gu
Nepal	: Pipla-mol
Common Name	: Thippili
Synonyms	: Kaman, Athi marunthu, Kolaiurike.
Botanical name	: Piper longum
Family	: Piperaceae
Parts used	: Immature berries (i.e., dried fruits)

Characters

Suvai	: Kaarppu
Thanmai	: Veppam
Pirivu	: Inippu

Chemical constituents

Volatile oil, Starch, resin, piperine, gum, Inorganic matter, fatty oil.

Therapeutic action:-

stimulant, carminative, alternative tonic, aphrodisiac.

Uses:-

“ஈளை யிரும லிரைப்புப் பசப்பிணிகள்
மாள வொழியாமல் வாட்டுமே- யாளுமுறை
பாங்கா யறிந்து செய்வீர் பண்டிதத்தைப் பண்டிதரே
வேங்கைவாய்ப் பான்கணை மெய்”

-தேரன் வெண்பா

It is used in Asthma, cough, disease of respiratory system

அன்னபேதி

Vernacular names

English	: Ferrous sulphate, Iron sulphate, Sulphates of Iron.
Tamil	: Annabedi
Malayalam	: Annabedi
Sanskrit	: Kasisa
Bengal	: Hira-Kas
Arab	: Zaje-Asfara
Hindi	: Haratutia
Gujarath	: Hara- Kasis
Common Name	: Annabedi.
Chemical Name	: Ferrous sulphate

Characters:-

Suvai	: Thuvarppu
Thanmai	: Veppam
Pirivu	: Kaarppu

Therapeutic action

Astringent, Emmenagogue, Antibilious, Antiperiodic

Uses

“அன்னபே திக்கே திக்த மாஞ்சுவை கஷாய மாம்லம்
சொன்னவீ ரியமே யுஷ்ணந் தொடர்கடு விபாக மாகுஞ்
துன்னிய கபமே வாதந் தொல்விடந் நேத்ர ரோகம்
பன்னுவெண் குஷ்டம் மேக ரணம்ஷயம் சுரமே பாண்டு”

- பதார்த்த பஞ்ச குண மஞ்சரி. பக்கம் -64

It is used in respiratory disease, vadha disease, vitligo, fever, Anaemia,
eye disease.

கொடி பவழம்

Vernacular Names

English	: Coral
Sanskrit	: Pravala, vidruma
Tamil	: Pavalam
Hindi	: Munga, Parvara
Malayalam	: Pavazham, Poalan,
German	: Korallian
French	: Corail
Telegu	: Pagadamu
Italy	: Corallo
Common name	: Kodi pavazham
Synonyms	: Thukir, thuppu, vithurumam, Senthendu malai
Chemical Name	: Corallium rubrum
Parts used	: Calcaneous shell, skeleton.

Chemical constituents

Organic matter, carbonate of lime, magnesium carbonates, magnesium oxide of iron. The red colour is due to its containing iron.

Therapeutic action

Antacid, Astringent, nervine tonic, diuretic, and laxative, antibilious.

Uses

“சுரதோடம் ஐயமுத் தோடசுரங் காசம்
அருசிகீ டத்தாலாம் ஆலம்- பெருவிந்து
நட்டம் அதிதாகம் நாவறட்சி போமோளியுங்
கிட்டும் பவழத்தாற் கேள்.”

- குணபாடம் பொருட்பண்பு நூல் தாது சீவ வகுப்பு

It is used in respiratory disease, fever, bronchitis, indigestion, dryness of the tongue, polydipsia, impotency.

Properties and uses

Its chief use is in cough, asthma, low fever, Head ache, giddiness, urinary disease, chronic bronchitis, and pulmonary tuberculosis.

- Indian materia medica –vol-III

A.K.Nadkarni.

ANNEXURE - II

BIO-CHEMICAL ANALYSIS OF ANNA PAVALA

CHENDURAM

PREPARATION OF THE EXTRACT

100mgs of chenduram is weighed accurately and placed into a clean beaker and added a few drops of conc. Hydrochloric acid and evaporated it well. After evaporation cooled the content and added a few drops of conc. Nitric acid and evaporated it well. After cooling the content add 20ml of distilled water and dissolved it well. Then it is transferred to 100ml volumetric flask and made up to 100ml with distilled water. Mix well. Filter it. Then it is taken for analysis.

Qualitative analysis:

S.No	Experiment	Observation	Inference
1.	TEST FOR CALCIUM 2ml of the above prepared extract is taken in a clean test tube. Add 2ml of 4% Ammonium oxalate solution is added to it	A White precipitate is formed	Indicates the presence of Calcium
2.	TEST FOR SULPHATE 2ml of the extract is added to 5% barium chloride solution	A white precipitate is formed	Indicates the presence of Sulphate.
3.	TEST FOR CHLORIDE The extract is treated with silver nitrate solution.	A white precipitate is formed	Indicates the presence of Chloride
4.	TEST FOR CARBONATE The substance is treated with concentrated HCL.	No brisk effervescence is formed	Absence of carbonate
5.	TEST FOR ZINC The extract is added with potassium ferro cyanide solution	No white precipitate is formed	Absence of Zinc

6.	TEST FOR IRON FERRIC: The extract is treated with glacial acid and potassium Ferro cyanide	Blue colour is formed	Indicates the presence of ferric Iron
7.	TEST OF IRON FERROUS: The extract is treated with concentrated Nitric acid and ammonium thiocynate	Blood red colour is formed	Indicates the presence of ferrous Iron.
8.	TEST FOR PHOSPHATE The extract is treated with ammonium Molybdate and concentrated nitric acid	No yellow precipitate is formed	Absence of phosphate
9.	TEST FOR ALBUMIN The extract is treated with Esbach's reagent	No yellow precipitate is formed	Absence of Albumin
10.	TEST FOR TANNIC ACID The extract is treated with Ferric chloride	No blue black precipitate is formed	Absence of Tannic acid
11.	TEST FOR UNSATURATION Potassium permanganate solution is added To the extract	It does not get decolourised	Absence of Unsaturated compound
12.	TEST FOR THE REDUCING SUGAR 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10drops of the extract and again boil it for 2 mts	No colour change occurs	Absence of Reducing sugar.
13.	TEST FOR AMINO ACID One or two drops of the extract is placed on a filter paper and dried it well after drying 1% Ninhydrin is sprayed over the same and Dried it well	No violet color it formed	Absence of Amino acid.

INFERENCE

The given sample of **ANNA PAVALA CHENDURAM** contains calcium, sulphate, chloride, ferric iron, ferrous iron.

BIO-CHEMICAL ANALYSIS OF KASHA CHOORANAM

PREPARATION OF THE EXTRACT

5gms of Chooranam was weighed accurately and placed in a 250ml clean beaker. Then 50ml distilled water is added and dissolved well. Then it is boiled well for about 10 minutes. It was cooled and filtered in a 100ml volumetric flask and then it is made up to 100ml with distilled water. This fluid is taken for analysis.

Qualitative analysis

S.No	Experiment	Observation	Inference
1.	TEST FOR CALCIUM 2ml of the above prepared extract is taken in a clean test tube. Add 2ml of 4% Ammonium oxalate solution is added to it.	No white precipitate is formed	Absence of Calcium
2.	TEST FOR SULPHATE 2ml of the extract is added to 5% barium chloride solution	No white precipitate is formed	Absence of Sulphate
3.	TEST FOR CHLORIDE The extract is treated with silver nitrate solution	No White precipitate is formed	Absence of carbonate
4.	TEST FOR CARBONATE The substance is treated with concentrated HCl	No brisk effervescence is formed	Absence of carbonate
5.	TEST FOR STARCH The extract is added with weak iodine solution	Blue colour is formed	Indicates the presence of starch

6.	TEST FOR IRON FERRIC: The extract is treated with glacial acetic acid and potassium Ferro cyanide	No blue colour is formed	Absence of ferric Iron
7.	TEST OF IRON FERROUS The extract is treated with concentrated Nitric acid and ammonium thio cynate	Blood red colour is formed	Indicates the presence of ferrous Iron.
8.	TEST FOR PHOSPHATE The extract is treated with ammonium Molybdate and concentrated nitric acid	No yellow precipitate is formed	Absence of phosphate
9.	TEST FOR ALBUMIN The extract is treated with Esbach's reagent.	No yellow precipitate is formed	Absence of Albumin
10.	TEST FOR TANNIC ACID The extract is treated with ferric chloride reagent.	No blue black precipitate if formed	Absence of Tannic acid
11.	TEST FOR UNSATURATION Potassium permanganate solution is added to the extract.	It gets decolourised	Indicates the presence of unsaturated compound
12	TEST FOR THE REDUCING SUGAR 5ml of Benedict's qualitative solution is taken in attest tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2mts.	No colour change occurs	Absence of Reducing sugar

13.	TEST FOR AMINO ACID One or two drops of the extracts is placed on a felted paper and dried it well after drying 1% Ninhydrin is sprayed over the same and dried it well.	Violet colour is formed	Indicated the presence of Amino acid
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INFERENCE

The given sample of **KASHA CHOORANAM** contains ferrous iron, starch, unsaturated compound, amino acid.

ANNEXURE - III

PHARMACOLOGICAL ANALYSIS

1. ANTI - HISTAMINIC EFFECT ON ANNA PAVALA CHENDURAM

AIM

To study the anti - histaminic effect of “**Anna Pavala chenduram**”.

PREPARATION OF THE TRIAL MEDICINE

1gm of “**Anna Pavala chenduram**” was taken and mixed with 5ml of water and 5ml of honey and filtered.

PROCEDURE

A guinea pig weighing about 350gm was starved for 48 hours and only water was allowed.

It was killed by stunning with a sharp blow on the head and cutting its throat to bleed to death. The abdomen was quickly opened and the viscera inspected and loops of intestine identified using the patch as a landmark. Then, the ileum was removed and placed in a shallow dish containing warm tyrode solution (37°C) and continuously aerated. The contents of the lumen of the ileum were washed and utmost care was taken to avoid any damage. It was cut into segments of 4 cm in a fully relaxed state and sutures were made with needle and tied on either side and the segment was suspended in an isolated organ bath. It was aerated by an oxygen tube immersed in tyrode solution. Drugs were given to study the inhibitory effect of histamine, induced contractions.

INFERENCE

The test drug “**Anna Pavala chenduram**” had moderate effect.

2. ANTI - HISTAMINIC EFFECT ON KASHA CHOORANAM

AIM

To study the anti - histaminic effect of “**Kasha Chooranam**”.

PREPARATION OF THE TRIAL MEDICINE

1gm of the “**Kasha Chooranam**” was taken and mixed with 5ml of water and 5ml of honey and filtered.

PROCEDURE

A guinea pig weighing about 350gm was starved for 48 hours and only water was allowed.

It was killed by stunning with a sharp blow on the head and cutting its throat to bleed to death. The abdomen was quickly opened and the viscera inspected and loops of intestine identified using the patch as a landmark. Then, the ileum was removed and placed in a shallow dish containing warm tyrode solution (37°C) and continuously aerated. The contents of the lumen of the ileum were washed and utmost care was taken to avoid any damage. It was cut into segments of 4 cm in a fully relaxed state and sutures were made with needle and tied on either side and the segment was suspended in an isolated organ bath. It was aerated by an oxygen tube immersed in tyrode solution. Drugs were given to study the inhibitory effect of histamine, induced contractions.

INFERENCE

The test drug “**Kasha Chooranam**” had moderate effect.

3. ANTI - SPASMODIC EFFECT ON ANNA PAVALA CHENDURAM

AIM

To study the anti - spasmodic effect on “**Anna Pavala chenduram**”

PREPARATION OF THE TRIAL MEDICINE

1gm of the “**Anna Pavala chenduram**” was taken and mixed with 5ml of water and 5ml of honey and filtered.

PROCEDURE

A rabbit weighing about 350 gm was starved for 48 hours and only water was allowed.

It was killed by stunning with a sharp blow on the head and cutting its throat to bleed to death. The abdomen was quickly opened and the viscera inspected and loops of intestine identified using the patch as a landmark. Then, the ileum was removed and placed in a shallow dish containing warm tyrode solution (37°C) and continuously aerated. The contents of the lumen of the ileum were washed and utmost care was taken to avoid any damage. It was cut into segments of 4 cm in a fully relaxed state and sutures were made with needle and tied on either side and the segment was suspended in an isolated organ bath. It was aerated by an oxygen tube immersed in tyrode solution. Drugs were given to study the inhibitory effect of Acetyl Choline.

INFERENCE

The test drug “**Anna Pavala chenduram**” had significant effect.

4. ANTI - SPASMODIC EFFECT ON KASHA CHOORANAM

AIM

To study the anti - spasmodic effect on “**Kasha Chooranam**”.

PREPARATION OF THE TRIAL MEDICINE

1gm of the “**Kasha Chooranam**” was taken and mixed with 5ml of water and 5ml of honey and filtered.

PROCEDURE

A rabbit weighing about 350 gm was starved for 48 hours and only water was allowed.

It was killed by stunning with a sharp blow on the head and cutting its throat to bleed to death. The abdomen was quickly opened and the viscera inspected and loops of intestine identified using the patch as a landmark. Then, the ileum was removed and placed in a shallow dish containing warm tyrode solution (37°C) and continuously aerated. The contents of the lumen of the ileum were washed and utmost care was taken to avoid any damage. It was cut into segments of 4 cm in a fully relaxed state and sutures were made with needle and tied on either side and the segment was suspended in an isolated organ bath. It was aerated by an oxygen tube immersed in tyrode solution. Drugs were given to study the inhibitory effect of Acetyl Choline.

INFERENCE

The test drug “**Kasha Chooranam**” had significant effect.

5. ACUTE ANTI-INFLAMMATORY STUDY ON

ANNA PAVALA CHENDURAM

BY HIND –PAW METHOD IN ALBINO RATS

AIM

To study the acute Anti-inflammatory effect of “**Anna Pavala Chenduram**” by Hind-paw Method in albino rats.

PREPARATION OF THE TEST DRUG

1gm of “**Anna Pavala Chenduram**” was dissolved in 10ml of water. A dose of 2ml was given to each rat. This 1ml contain 100mg of the test drug.

PROCEDURE

Six healthy albino rats of both sexes each weighing between 100 - 150mg were taken and divided in to these groups, each consisting of 2 rats.

First group was kept on control by giving distilled water of 1ml/100gm of body weight. The second group was given. Ibuprofen at dose of 20mg/100gm of body weight. The third group received the test drug “**Anna Pavala Chenduram**” of 20mg/100gm of body weight.

Before administration of test drug, the hind-paw volume of all rats was measured.

This was done by dipping the hind-paw (upto tibio – tarsal Junction) into a mercury plethysmograph. While dipping the hind – paw by pulling the syringe piston, the level of mercury in the center small tube was made to coincide with red marking and reading was noted from the plethysmograph.

Soon after the measurement the drugs were administered orally. One hour later a sub-cutaneous injection of 0.1ml of 1% (W/V) carrageen in water was made into plants surface of both hind –paw of each rat. Three hours after

carrageen injection, the hind-paw volume was measured once again. The difference between the initial and final volume was calculated and compared.

The method is more suitable for studying the anti-inflammatory activity in acute inflammation. The values are given in the table. Effect of **“Anna Pavala Chenduram”** in acute-inflammatory study.

INFERENCE

The medicine **“Anna Pavala Chenduram”** has significant acute anti-inflammatory effect.

Study of Acute Anti-Inflammatory by Hind paw method Using plethysmograph Using the Drug on Anna pavala chenduram

S. No	Name of Drugs/Groups	Dose/100 gram body weight	Initial Reading average	Final reading average	Mean difference	Percentage inflammation	Percentage inhibition
1	Control	2ml	0.55	1.4	0.85	100	Nil
2.	Std(Ibu brufen)	20mg	0.55	0.85	0.3	35.2	64.8
3.	Anna pavala chenduram	40mg	0.87	1.15	0.28	32.9	67.1
Singnificant - Action							

6. ACUTE ANTI-INFLAMMATORY STUDY ON KASHA CHOORANAM BY HIND –PAW METHOD IN ALBINO RATS

AIM

To study the acute Anti-inflammatory effect of “**Kasha Chooranam**” by Hind-paw Method in albino rats.

PREPARATION OF THE TEST DRUG

1gm of “**Kasha Chooranam**” was dissolved in 10ml of water. A dose of 2ml was given to each rat. This 1ml contain 100mg of the test drug.

PROCEDURE

Six healthy albino rats of both sexes each weighing between 100 - 150mg were taken and divided in to these groups, each consisting of 2 rats.

First group was kept on control by giving distilled water of 1ml/100gm of body weight. The second group was given. Ibuprofen at dose of 20mg/100gm of body weight. The third group received the test drug “**Kasha Chooranam**” of 20mg/100gm of body weight.

Before administration of test drug, the hind-paw volume of all rats was measured.

This was done by dipping the hind-paw (upto tibio – tarsal Junction) into a mercury plethysmograph. While dipping the hind – paw by pulling the syringe piston, the level of mercury in the center small tube was made to coincide with red marking and reading was noted from the plethysmograph.

Soon after the measurement the drugs were administered orally. One hour later a sub-cutaneous injection of 0.1ml of 1% (W/V) carrageen in water was made into plants surface of both hind –paw of each rat. Three hours after

carrageen injection, the hind-paw volume was measured once again. The difference between the initial and final volume was calculated and compared.

The method is more suitable for studying the anti-inflammatory activity in acute inflammation. The values are given in the table. Effect of “**Kasha Chooranam**” in acute-inflammatory study.

INFERENCE

The medicine “**Kasha Chooranam**” has significant acute Anti-inflammatory effect.

Study of Acute Anti-Inflammatory by Hind paw method Using plethysmograph Using the Drugs on Kasha chooranam

S. No	Name of Drugs/Groups	Dose/100 gram body weight	Initial Reading average	Final reading average	Mean difference	Percentage inflammation	Percentage inhibition
1	Control	2ml	0.55	1.4	0.85	100	Nil
2.	Std (Ibuprofen)	20mg	0.55	0.85	0.3	35.2	64.8
3.	Kasha chooranam	200mg	0.45	0.8	0.35	41.17	58.8
Significant - Action							

**7. CHRONIC – ANTI – INFLAMMATORY STUDY ON
ANNA PAVALA CHENDURAM
BY COTTON PELLETS GRANULOMA METHOD
IN ALBINO RATS**

AIM

To study the chronic anti-inflammatory activity of the drug, “**Anna Pavala Chenduram**” in the rats by cotton pellets implantation (granuloma) methods in albino rats.

PREPARATION OF THE TEST DRUG

1gm of “**Anna Pavala Chenduram**” was mixed with dissolved in 10ml of distilled water. A dose 1ml was given to each rat. This 1ml contains 100mg of test drug.

PROCEDURE

Cotton pellets each weighing 10mg were prepared and sterilized in the autoclave for about one hour under 15 lbs atmospheric pressure, 6 albino rats weighing between 100-200gm were selected and divided into 3 groups each containing 2 rats. Each rat was anesthetized with ether and cotton pellets were implanted subcutaneously in the groin of two in each side.

From the day of implantation a group of animals received “**Anna Pavala Chenduram**” in a dose of 100mg/100gm of body weight. The control group of animals received distilled water 1ml/100gm of body weight.

The standard group of animals received distilled water 1ml/100gm of body weight. The standard group of animals received Ibuprofen in a dose of 20mg/100gm of body weight.

On the eighth day the rats were sacrificed and the pellets were removed and weighed. They were put in an incubator at 60⁰ – 80⁰C and then they were weighed.

The weight of the granulation tissue formed is the difference between the weight and dry weight. The results of the control standards and test group were compared and the results were calculated. Effect of “**Anna Pavala Chenduram**” in chronic anti-inflammatory study.

INFERENCE

The medicine “**Anna Pavala Chenduram**” has significant chronic Anti-inflammatory effect.

Study of Chronic Anti-Inflammatory effect by Cotton Pellet method Using the Drugs of Anna pavala chenduram.

S. No	Name of Drugs/Groups	Dose/100 gram body weight	Pellet weight	Pellet weight of the Granuloma of drugs	Mean difference	Percentage inflammation	Percentage inhibition
1	Control	1ml	10mg	250mg	240mg	100	Nil
2.	Std (Ibu brufen)	20mg	10mg	55mg	45mg	22	78
3.	Anna pavala chenduram	40mg	10mg	98mg	88mg	40	60
Singnificant - Action							

**8. CHRONIC – ANTI – INFLAMMATORY STUDY ON
KASHA CHOORANAM
BY COTTON PELLETS GRANULOMA METHOD
IN ALBINO RATS**

AIM

To study the chronic anti-inflammatory activity of the drug, “**Kasha Chooranam**” in the rats by cotton pellets implantation (granuloma) methods in albino rats.

PREPARATION OF THE TEST DRUG

1gm of “**Kasha Chooranam**” was mixed with dissolved in 10ml of distilled water. A dose 1ml was given to each rat. This 1ml contains 100mg of test drug.

PROCEDURE

Cotton pellets each weighing 10mg were prepared and sterilized in the autoclave for about one hour under 15 lbs atmospheric pressure, 6 albino rats weighing between 100-200gm were selected and divided into 3 groups each containing 2 rats. Each rat was anesthetized with ether and cotton pellets were implanted subcutaneously in the groin of two in each side.

From the day of implantation a group of animals received “**Kasha Chooranam**” in a dose of 100mg/100gm of body weight. The control group of animals received distilled water 1ml/100gm of body weight.

The standard group of animals received distilled water 1ml/100gm of body weight. The standard group of animals received Ibuprofen in a dose of 20mg/100gm of body weight.

On the eighth day the rats were sacrificed and the pellets were removed and weighed. They were put in an incubator at 60⁰ – 80⁰C and then they were weighed.

The weight of the granulation tissue formed is the difference between the weight and dry weight. The results of the control standards and test group were compared and the results were calculated. Effect of “**Kasha Chooranam**” in chronic anti-inflammatory study.

INFERENCE

The medicine “**Kasha Chooranam**” has significant chronic Anti-inflammatory effect.

Study of Chronic Anti-Inflammatory effect by Cotton Pellet method Using the Drugs of Kasha chooranam

S. No	Name of Drugs/Groups	Dose/100 gram body weight	Pellet weight	Pellet weight of the Granuloma of drugs	Mean difference	Percentage inflammation	Percentage inhibition
1	Control	10ml	10mg	250mg	240mg	100	Nil
2.	Std	20mg	10mg	55mg	45mg	22	78
3.	Kasha chooranam	100mg	10mg	100mg	90mg	40	60
Singnificant - Action							

ANNEXURE - IV
MICROBIOLOGICAL STUDIES
ANTI MICROBIAL STUDY OF KASHA CHOORANAM
AND ANNA PAVALA CHENDURAM

AIM

To study the anti microbial action of “**Kasha Chooranam and Anna Pavala Chenduram**”

PROCEDURE

To prepare the chooranam and chenduram 40 mg concentration of the drug, 4 grams of the drug was dissolved in 1 ml of sterile distilled water and from this master solution 40 micro litre was loaded on the disc.

PREPARATIONS OF STANDARD STRAINS

Standard laboratory referral strains such were initially grown in nutrient agar and maintained at 37°C.

Before antibacterial testing each strain was inoculated in 5ml of Brain Heart Infusion broth (B.H.I) and incubated at 37°C for 30 minutes.

ANTIMICROBIAL ACTIVITY TESTING BY KIRBY - BAUER DISC DIFFUSION METHOD

For antimicrobial activity 90mm petriplates with Muller Hinton. Agar (M.H.A) was used, for each organism, one M.H.A plate was used. The organisms grown in B.H.I Broth with 0.5 Mcfarland turbidity standard was poured on the M.H.A plate and allowed to spread uniformly. The excess broth was drained aseptically.

The disc which contains 40mg concentration of the drug was placed in M.H.A and incubated at 37°C for 24 hours.

INTERPRETATION

Readings were taken after 24 hours of incubation. The inhibitory zone diameter was measured in millimeter scale.

RESULT

Kasha Chooranam and Anna Pavala Chenduram was compared with standard antibiotics. The medicine was well sensitive against **staphylococcus aureus** and **pseudomonas aeruginosa**.

ANNEXURE - V

PROFORMA OF CASE SHEET

GOVERNMENT SIDDHA MEDICAL COLLEGE AND HOSPITAL

DEPARTMENT OF POST GRADUATE POTHU MARUTHUVAM

PALAYAMKOTTAI - TIRUNELVELI - 627002

CASE SHEET PROFORMA FOR “SWASAKASAM” - (IP)

Ward :	Occupation :
I.P. No :	Income :
Bed No :	Nationality :
Name :	Religion :
Age/Sex :	Date of admission :
Address :	Date of discharge :
	Result :
	Diagnosis : SWASAKASAM
	Total No of days Treated :
	Medical officer :

COMPLAINTS AND DURATION:

HISTORY OF PRESENT ILLNESS:

HISTORY OF PAST ILLNESS:

PERSONAL HISTORY:

FAMILY HISTORY:

HABITS:

OCCUPATIONAL HISTORY:

GENERAL EXAMINATION:

Consciousness :
 Decubitus :
 Nourishment :
 Anaemia :
 Jaundice :
 Clubbing :
 Cyanosis :
 Lymphadenopathy :
 JVP :
 Pedal Oedema :
 Congenital anomaly :

VITAL SIGNS :

Temperature :
 Pulse Rate :
 Respiratory Rate :
 Blood Pressure :

❖ SIDDHA ASPECTS**NILAM :**

Kuringi :
 Mullai :
 Marutham :
 Neithal :
 Palai :

PARUVAKALAM :

Kaar :
 Koothir :
 Munpani :
 Pinpani :
 Ilavenil :
 Muthuvenil :

SIRUPOLUDHU:

Vaigarai :
 Pagal :
 Nanpagal :
 Pirpagal :
 Maalai :
 Yamam :

IMPORIGAL & IMPULANGAL

Mei :
 Vai :
 Kann :
 Mookku :
 Sevi :

KANMENTHIRIYAM & KANMAVIDAYAM

Kai :
 Kaal :
 Vai :
 Eruvai :
 Karuva :

KOSAM:

Annamayakosam :

(Digestive System)

Pranamayakosam :

(Respiratory System)

Manomayakosam :

(Cardio Vascular System)

Gnanamayakosam :

(Central Nervous System)

Aananthamayakosam :

(Reproductive System)

UYIR THATHUKKAL:**VATHAM**

Pranan :

Abanan :

Viyanan :

Uthanan :

Samanan :

Nagan :

Koorman :

Kirugaran :

Devathathan :

Dhananjayan :

PITHAM

Anarpitham :

Ranjagam :

Sathagam :

Alosagam :

Prasagam :

KABAM

Avalambagam	:
Kilethagam	:
Tharpagam	:
Pothagam	:
Sandhigam	:

UDAL KATTUGAL

Saaram	:
Senneer	:
Oon	:
Enbu	:
Kozhuppu	:
Moolai	:
Sukkilam / Suronitham	:

ENVAGAI THERVUGAL

Naadi	:
Sparism	:
Naa	:
Niram	:
Mozhi	:
Vizhi	:
Malam	:
Moothiram	:

(a) Neerkuri:

i. Niram	:
ii. Manam	:
iii. Edai	:
iv. Nurai	:
v. Engal	:

(b) Neikuri:

❖ MODERN ASPECTS

I. Inspection

1. Trachea :
2. Chest wall symmetry :
3. Chest wall abnormality :
4. Harrison's sulcus :
5. Apical impulse :
6. Pulsatile swelling :
7. Intercostal Muscle Wasting:
8. Drooping of shoulder :
9. Intercostal bulging :
10. Respiratory Movement :
11. Measurements – AP :
- Transverse :
12. Suprasternal pulsation :
13. Carotid pulse :
14. Jugular venous pulsation :

II. Palpation

1. Tracheal position (Tracheal sign) :
2. Apical impulse :
3. Respiratory Movements :
4. Local tenderness :
5. Tactile Fremitus :
6. Vocal Fremitus :

III. Percussion

1. Abnormal Dulness :
 - i. Hyper Resonance :
 - ii. Dullness :
 - iii. Stony Dullness :
 - iv. Tidal percussion :
 - v. Ellie's 'S' shaped curve :

- vi. Straight line dullness :
- vii. Shifting dullness :
- viii. Succussion splash :
- 2. Traube's space :
- 3. Upper border of liver dullness :
- 4. Cardiac border :
- 5. Kronig's isthmus sign :

Auscultation

- 1. Breath sounds
 - NVBS
 - Bronchial breathing
 - I. Tubular breathing
 - II. Cavernous breathing
 - III. Amphoric
- 2. Adventitious sounds
 - Wheeze continuous
 - Crackles interrupted
 - Pleural rub
- 3. Vocal Resonance
 - Aegophony
 - Bronchophony
 - Whispering pectorilology

IV. Other systemic Examination

- Cardio Vascular System :
- Gastro Intestinal System :
- Central Nervous System :
- Musculo Skeletal System :

VI. Lab Investigations

Blood

BT

AT

TC :

DC :

ESR :

Hb :

Blood Sugar(R) :

Blood Urea :

Serum Cholesterol :

Urine

Albumin :

Sugar :

Deposits :

Motion

Ova :

Cyst :

VII. Other Investigations

a) Mantoux Test :

b) Sputum AFB :

c) X-Ray Chest PA View :

d) Pulmonary Function Test :

e) Arterial Blood Gases

and oximetry :

f) Absolute Eosinophil count :

Treatment:

Diet :

Advice :

**GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,
PALAYAMKOTTAI**

DEPARTMENT OF POST GRADUATE - POTHU MARUTHUVAM

DISCHARGE CASE SHEET - 'SWASA KASAM'

I.P.No :	Occupation :	
Bed No :	Income :	
Ward No :	Nationality :	
Name :	Religion :	
Age/Sex :	Date of Admission :	
Address ` :	Date of Discharge :	
	No.of.Days treated :	
	Diagnosis :	'SWASA KASAM'
	Result :	
	Medical officer :	

Clinical Features:

S.No	Signs and Symptoms	During Admission	During Discharge
1.	Running Nose		
2.	Sneezing		
3.	Difficulty in breathing		
4.	Cough with expectoration		
5.	Tightness of chest		
6.	Clubbing		
7.	Cyanosis		
8.	Sweating		
9.	Tachycardia		
10.	Fever		
11.	Eosinophil		
12.	Peak expiratory flow meter rate		

O.P No during Follow up :

No. of Days I.P Treated :

Total No.of Days Treated :

PROFORMA OF CASE SHEET
GOVERNMENT SIDDHA MEDICAL COLLEGE AND HOSPITAL
DEPARTMENT OF POST GRADUATE POTHU MARUTHUVAM
PALAYAMKOTTAI - TIRUNELVELI - 627002

CASE SHEET PROFORMA FOR “SWASAKASAM” - O.P

O.P. No :	Occupation	:
Name :	Income	:
Age/sex :	Treatment Starting Date	:
Address :	End of the Treatment Date	:
	Total No. of Days Treated	:
	Result	:
	Diagnosis	: Swasakasam
	Medical Officer	:

COMPLAINTS AND DURATION

1. Running nose :
 2. Sneezing :
 3. Difficulty in breathing :
 4. Cough with expectoration :
 5. Tightness of chest :
 6. Fever :
 7. Flatulence :
 8. Excessive salivation :
 9. Throat irritation :
 10. Sweating :
 11. Tachycardia :
 12. Sputum :
- DURATION OF ILLNESS :
- PAST HISTORY :
- OCCUPATIONAL HISTORY :

GENERAL EXAMINATION

Consciousness	:	Temperature	:
Decubitus	:	Pulse rate	:
Nourishment	:	Heart rate	:
Anaemia	:	Respiratory rate	:
Jaundice	:	Blood Pressure	:
Clubbing	:		
Cyanosis	:		
Lymphadenopathy	:		
Pedaloedema	:		
JVP	:		

ENVAGAI THERVUGAL

Naadi	:
Sparisam	:
Naa	:
Niram	:
Mozhi	:
Vizhi	:
Malam	:
Moothiram	:

a) Neerkuri

- i. Niram :
- ii. Manam :
- iii. Edai :
- iv. Nurai :
- v. Enjal :

b) Neikuri :

Lab Investigations:

Blood	BT	AT
TC	:	
DC	:	
ESR	:	
Hb	:	
Blood Sugar(R)	:	
Blood Urea	:	
Serum Cholesterol	:	
Urine		
Albumin	:	
Sugar	:	
Deposits	:	
Motion		
Ova	:	
Cyst	:	

OTHER INVESTIGATIONS:

- a. Mantoux Test :
- b. Sputum AFB :
- c. X - Ray Chest PA view :
- d) Pulmonary Function Test :
- e) Arterial Blood Gases
and oximetry :
- f) Absolute Eosinophil count :

EXAMINATION OF RESPIRATORY SYSTEM

Trachea	:
Respiratory Movements	:
Auscultation	:
Breath Sounds	:
NVBS	:
Bronchial breathing	:

I. Tubular breathing :
II. Cavernous breathing :
III. Amphoric :
Added sounds :
Peak Expiratory Flow Meter Rate :

OTHER SYSTEMIC EXAMINATION

Cardio Vascular System :
Gastro Intestinal System :
Central Nervous System :
Musculo Skeletal System :

Treatment :

Diet :

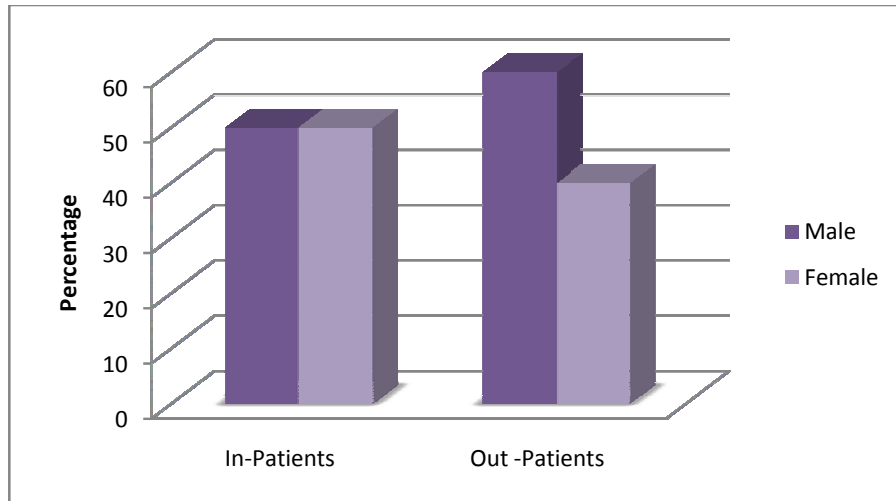
Advice :

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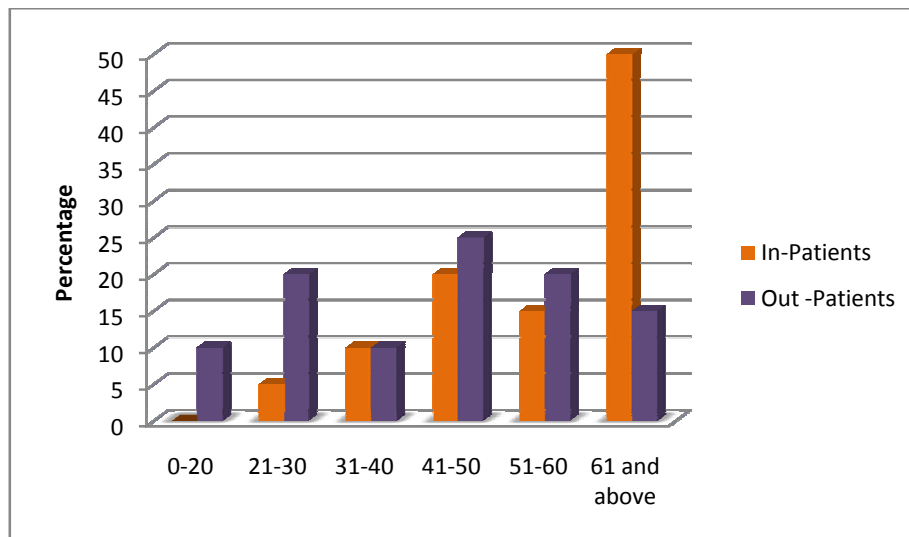
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- S.P.Ramachandran.
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- S.Krishanrao.
10. Siddha maruthuvanga churukkam.
- Dr.C.S.Uthamarayan.
- 11.Thotrakirama Araichium Siddha Maruthuva varalarum.
- Dr.C.S.Uthamarayan.
12. Gunapadam- mooligai vaguppu.
-Dr.S.Murugesu mudhaliyar.
13. Gunapadam- Thathu and Jeeva vaguppu.
-Dr.R.Thiyagarajan C.I.M.
14. Siddha Maruthuvam sirappu.
-Dr.R.Thiyagarajan C.I.M.

15. Maruthuva Thavara Iyal.
-Dr.Somasundaram .
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17. Thanvandhiri Vaidhyam- I volume.
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31. Davidson's Principle and Practice of Medicine.
32. Manual of Practical Medicine.
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33. Hutchison's Clinical Methods.
34. Pharmacology and Pharmacotherapeutics.
- Dr. R.S. Satoskar , B.Sc .,M.B.B.S., Ph.D.,

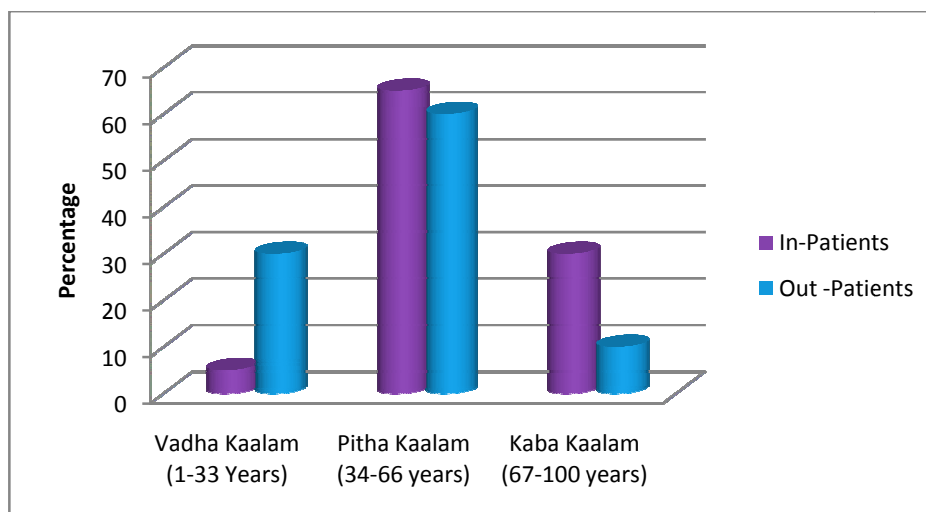
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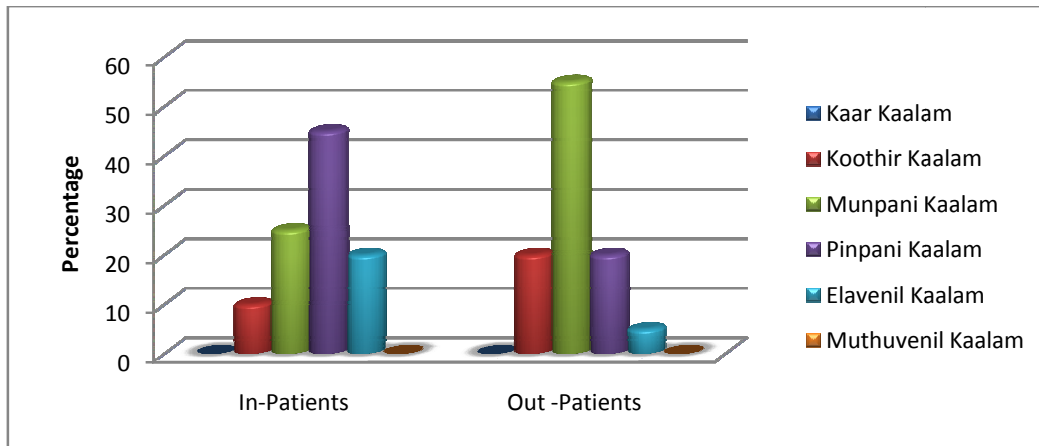
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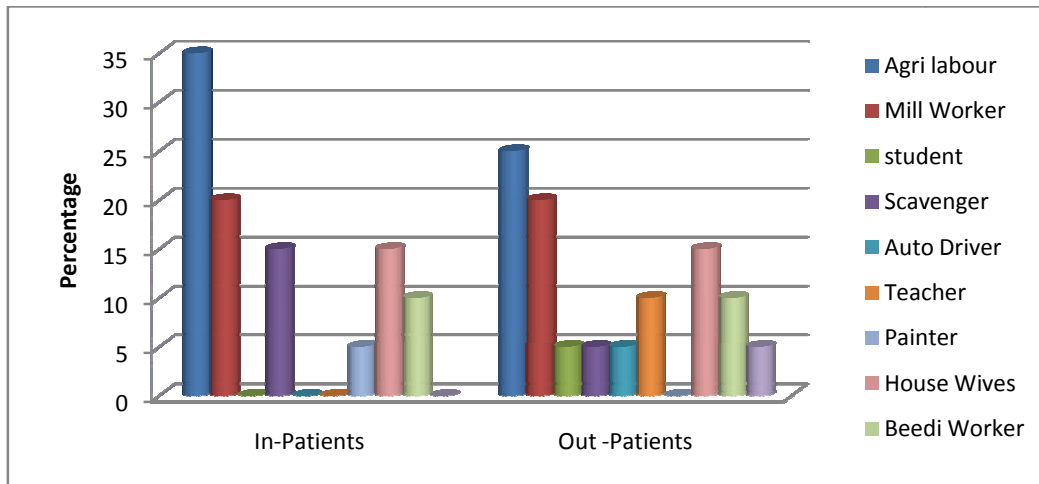
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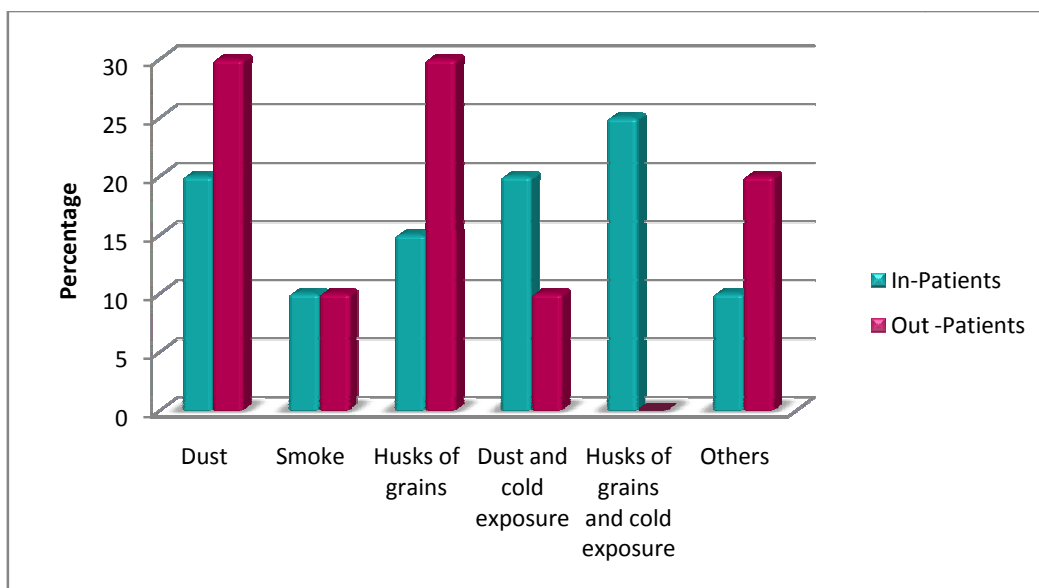
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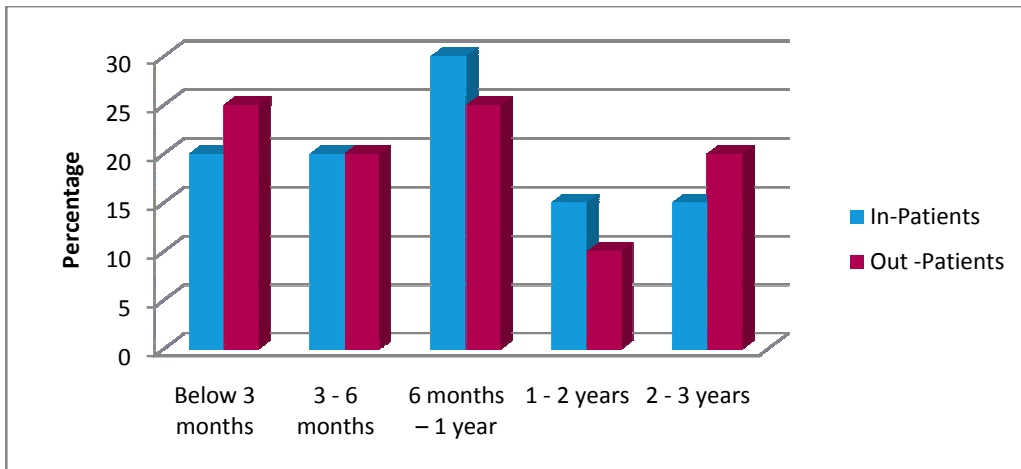
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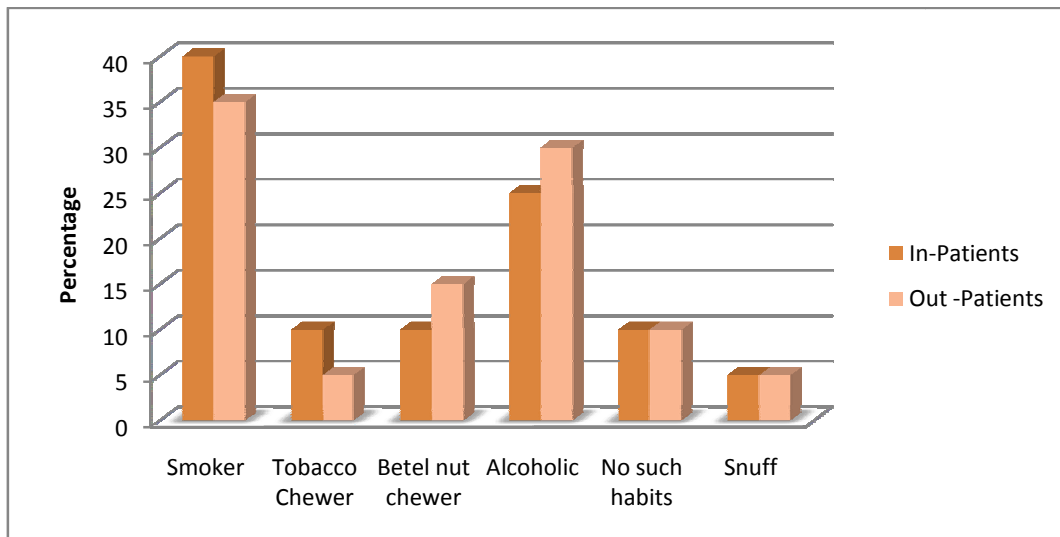
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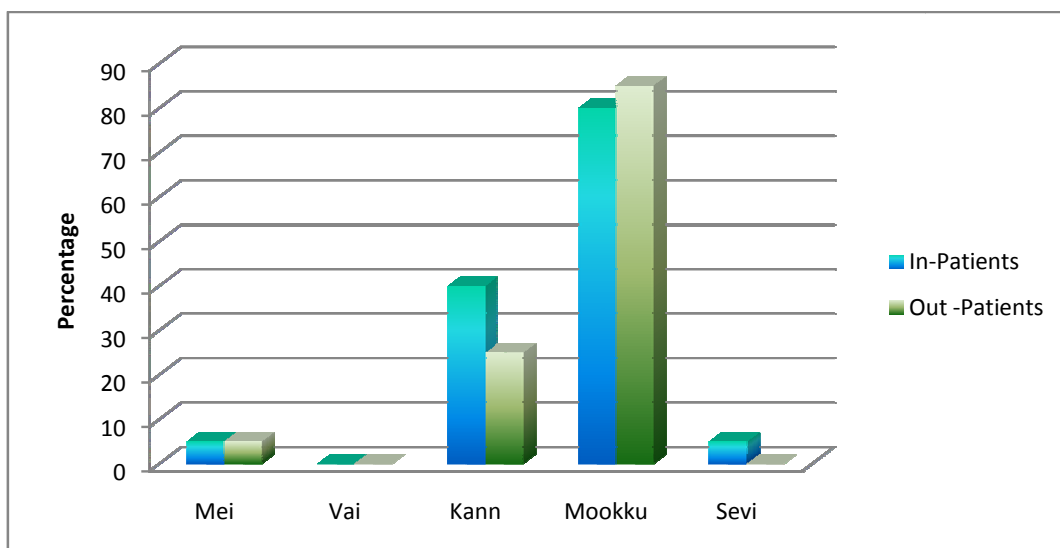
Duration of illness



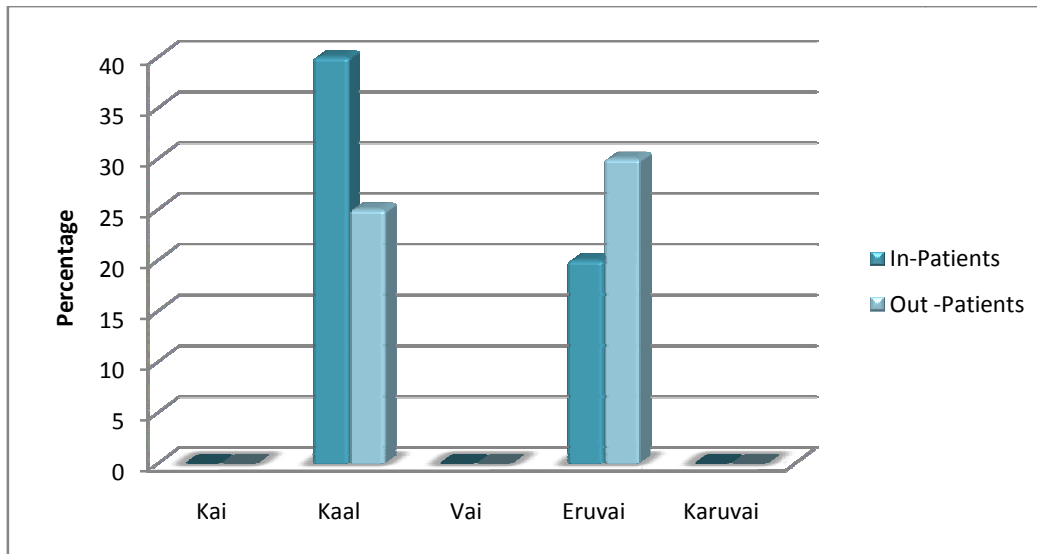
Habits



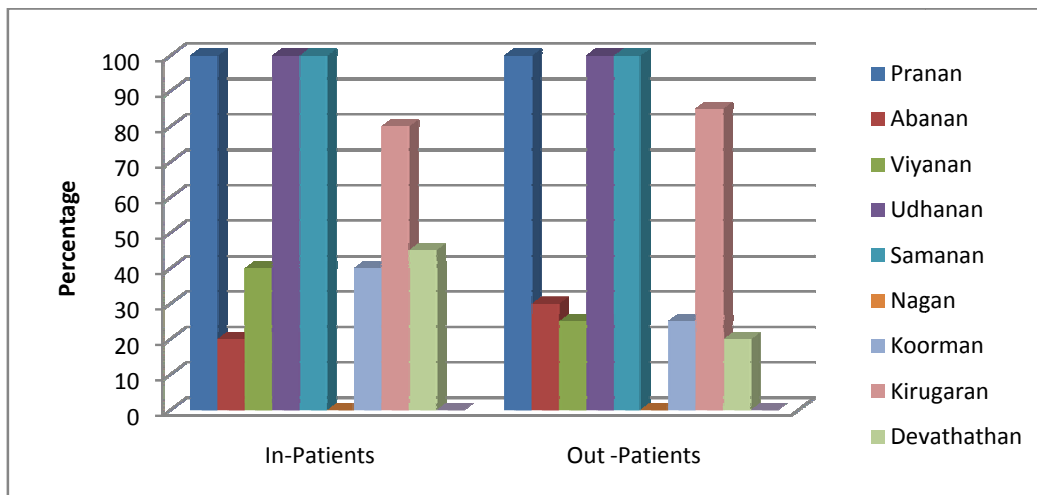
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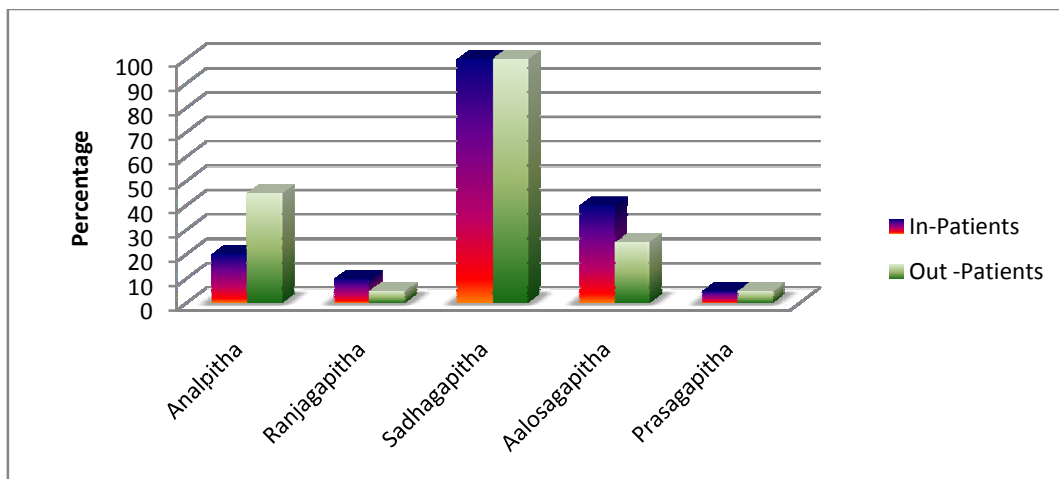
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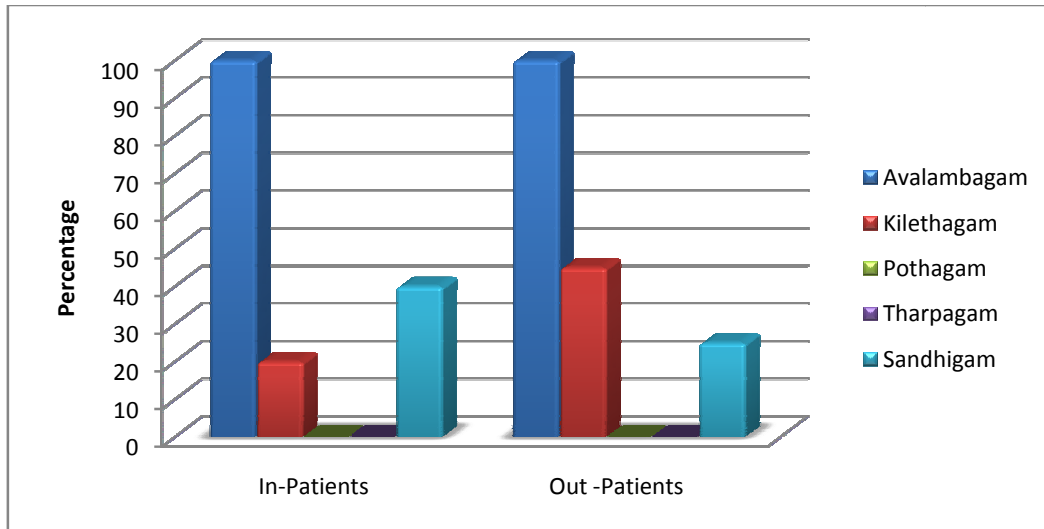
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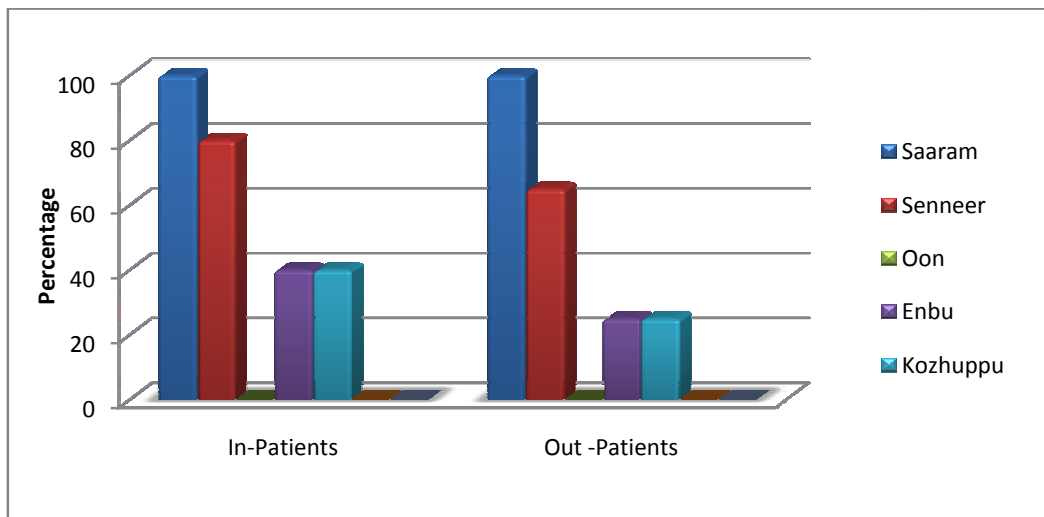
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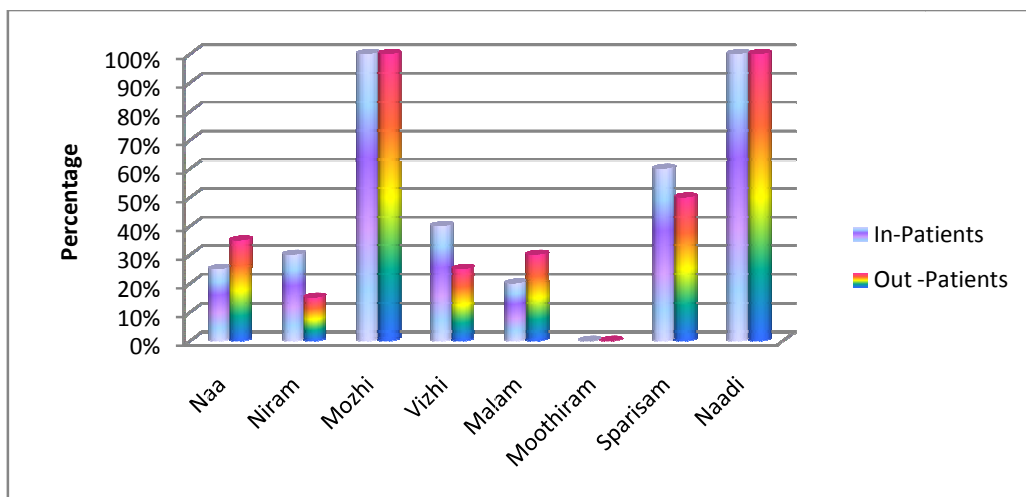
Kabam



Ezhu Udal Kattugal



Envagai Thervugal



Result

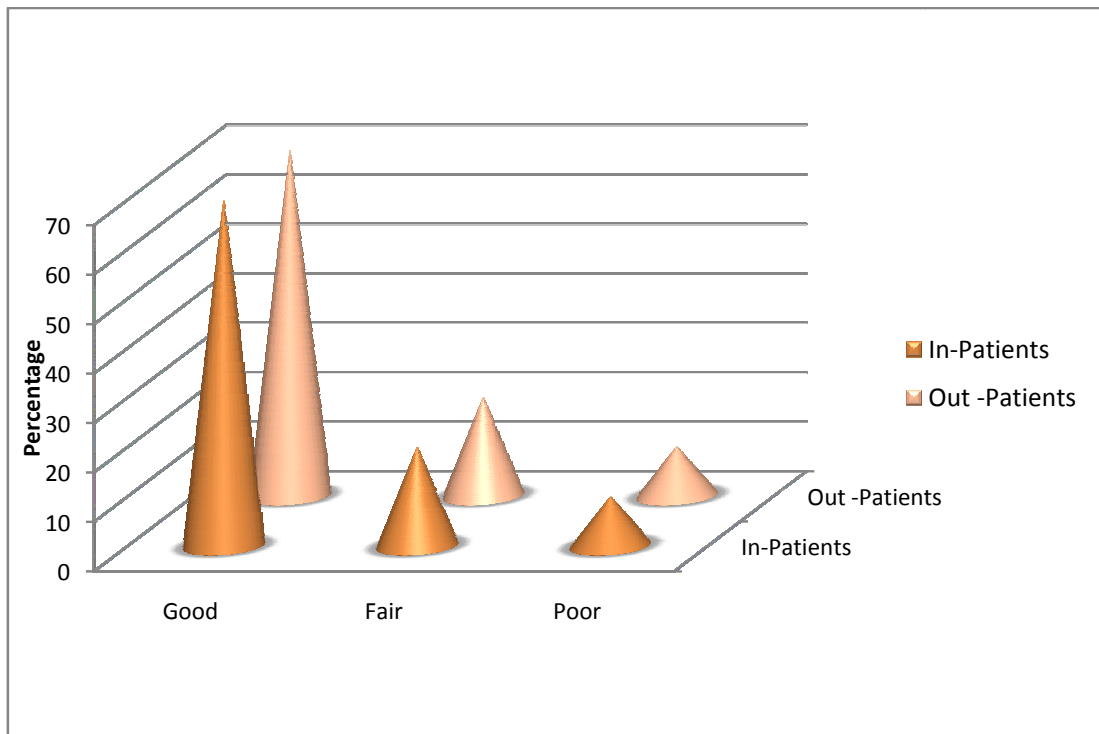


Table - 27- A

S.No	I.P.No	Name	Age	Sex	Habit	Duration of illness in months	Occupation	Etiological Factor (allergen)	Date of Admission	Date of Discharge	No. of days treated			Blood sugar	Blood Urea	Serum Cholesterol	X-Ray Chest PA view	Peak flow Meter Reading L/min		Asthmatic episodes / week		Family History	Result
											As in IP	As in OP	Total					BT	AT	BT	AT		
1	3012	Poovalingam	65	M	S	18	Mill	S	11/12/2007	5/1/2008	26	-	26	107	26	133	Normal	120	310	8	2	-	Good
2	3021	Arumugam	70	M	-	6	Painter	H.G	12/12/2007	8/1/2008	28	-	28	98	22	136	Normal	180	350	5	1	-	Good
3	1060	Nagarajan	69	M	S & A	6	Agri	H.G & CE	26/04/08	15/05/08	20	-	20	72	18	160	Normal	170	360	4	1	-	Good
4	170	Subramanian	75	M	S	24	Scavenger	Dust	21/01/08	8/2/2008	19	-	19	76	26	162	Normal	90	230	7	3	-	Fair
5	229	Arumugam	65	M	S	4	Beedi	-	26/01/08	15/02/08	21	-	21	105	20	142	Normal	200	380	3	1	-	Good
6	196	Kanthaiya	62	M	S	8	Mill	Dust	24/01/08	13/02/08	21	-	21	98	21	178	Normal	150	350	5	2	-	Good
7	178	Vellasamy	65	M	S	24	Mill	D & C	23/01/08	15/02/08	24	-	24	82	14	170	Normal	80	170	9	6	+	Poor
8	322	Mariyappan	38	M	A	6	Agri Labour	D & C	4/2/2008	15/02/08	12	-	12	88	16	152	Normal	150	330	8	2	-	Good
9	184	Savithiri	48	F	-	3	Scavenger	H.G & CE	23/01/08	15/02/08	18	-	18	87	20	173	Normal	170	360	7	2	-	Good
10	788	Thamayanthi	67	F	T	18	House Wife	H.G	26/03/08	15/04/08	21	10	31	92	24	185	Normal	110	260	8	4	-	Fair
11	823	Pappa	30	F	-	5	Agri Labour	Dust	28/03/08	17/04/08	21	-	21	90	14	139	Normal	150	360	8	3	-	Good
12	792	Ramaya	60	M	S & A	12	Agri Labour	D & C	26/03/08	17/04/08	19	-	19	86	18	150	Normal	90	250	5	2	+	Fair
13	659	Mani vel	73	M	A	9	Agri Labour	H.G & CE	10/3/2008	20/03/08	11	23	34	98	18	152	Normal	100	270	7	3	-	Fair
14	722	Iyamperumal	50	M	-	4	Agri Labour	D & C	17/03/08	1/4/2008	16	20	36	102	15	127	Normal	160	370	6	2	+	Good
15	778	Paechiyammal	48	F	B	5	Agri Labour	Dust	25/03/08	7/4/2008	14	20	34	95	20	170	Normal	180	350	4	1	-	Good
16	859	Ponnammal	42	F	-	1	House Wife	H.G & CE	3/4/2008	17/04/08	15	-	15	300	20	198	Normal	190	370	3	1	+	Good
17	1051	Kamalam	55	F	T	2	Scavenger	-	24/04/08	13/05/08	20	-	20	100	18	126	Normal	150	360	5	2	-	Good
18	991	Avadaiyammal	40	F	B	2	House Wife	H.G	17/04/08	7/5/2008	21	10	31	90	16	152	Normal	110	300	4	1	-	Good
19	730	Arumugam	55	F	-	7	Beedi	H.G & CE	18/03/08	8/4/2008	22	20	42	101	21	147	Normal	120	320	8	2	-	Good
20	257	Prammanagam Pillai	68	M	-	36	Mill	S	30/01/08	10/2/2008	12	-	12	82	20	130	Bronchitis	70	150	8	5	+	Poor

H.G - Husks of Grains
D & C - Dust & Cold Exposure
H.G & CE - Husks of Grains & Cold Exposure
S - Smoker

T - Tobacco Chewer
B - Betelnut Chewer
A - Alcoholic
A & S - Alcoholic & Smoker

(-) Negative
(+) Positive

Table - 27- B

S. No	IP. No	BLOOD INVESTIGATION														URINE ANALYSIS						MOTION			
		BT				HB %	AT				HB %	BT		AT		BT			AT						
		TC	DC				TC	DC				ESR	ESR	Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst		
			P	L	E			P	L	E														1/2	1
1	3012	7600	52	32	4	73	9000	52	34	2	78	2	7	1	3	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
2	3021	8600	60	32	8	71	9800	62	37	3	80	20	42	4	8	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
3	1060	9000	50	47	3	74	10000	58	41	1	79	4	8	1	3	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
4	170	8300	50	38	12	76	9500	54	42	4	71	14	18	4	6	Nil	Nil	few epi	NIL	NIL	NAD	NIL	NIL	NIL	NIL
5	229	8100	70	27	3	81	9200	67	32	1	84	6	14	2	5	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
6	196	8100	69	28	3	72	9700	72	27	1	75	10	20	2	4	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
7	178	9000	60	34	6	71	10300	56	39	5	73	5	10	2	7	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
8	322	10000	68	29	3	78	10300	66	33	1	85	10	22	2	5	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
9	184	9800	60	33	7	71	10700	58	40	2	76	5	10	1	3	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
10	788	8500	58	40	12	72	9800	60	35	5	78	7	14	4	7	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
11	823	9200	50	42	8	70	10400	52	46	2	75	8	15	2	4	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
12	792	8600	48	48	4	73	10000	49	49	2	80	12	20	5	8	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
13	655	9800	66	30	4	76	10300	64	34	2	81	7	15	4	8	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
14	722	9400	67	30	3	52	10200	68	31	1	68	10	20	3	5	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
15	778	9000	58	38	4	74	9800	60	38	2	80	5	10	2	4	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
16	859	9500	53	43	4	75	10500	52	47	1	81	10	20	2	5	Nil	++	1-2 pus cells	NIL	NIL	NAD	NIL	NIL	NIL	NIL
17	1051	8900	65	30	5	72	9600	66	32	2	78	5	10	1	3	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL

18	991	9200	68	28	4	60	10100	70	29	1	71	2	7	1	3	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
19	730	8800	56	38	6	70	9600	58	40	2	81	10	20	4	6	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
20	257	8800	60	34	6	73	9500	58	38	4	79	17	38	8	15	Nil	Nil	1-2 epi	NIL	NIL	NAD	NIL	NIL	NIL	NIL

Table - 27- C

S. No	OP. No	Name	Age	Sex	Duration of Illness in months	Habit	Etiological Factor	Occupation	No. of days treated	Blood Sugar	Blood Urea	Serum Cholestrol	X-Ray chest PA view	Peak flow Meter readings L/min		Asthmatic episodes / week		Family History	Results
														BT	AT	BT	AT		
1	6950	Pandiammal	59	F	5	B	Dust	House wife	60	101	26	191	Normal	120	300	5	1	-	Good
2	1505	Malathi	27	F	2	-	D & C	Nurse	52	76	15	192	Normal	180	320	4	1	+	Good
3	74450	Muthu Mari	21	F	3	-	Dust	Mill	53	81	17	170	Normal	150	300	6	1	-	Good
4	73049	Subbu	50	F	18	-	D & C	Mill	48	100	18	180	Normal	90	280	6	3	-	Fair
5	9379	Seetha lakshmi	45	F	12	Snuff	H.G	Agri labour	46	79	12	161	Normal	80	300	4	1	+	Good
6	72140	Arumugam	55	M	24	S & A	S	Beedi	51	96	24	185	Bronchitis	70	140	8	6	-	Poor
7	698	Subramanian	75	M	24	S	Dust	Scavenger	25	76	26	162	Normal	90	210	7	3	-	Fair
8	18925	Revathi	36	F	12	-	H.G	Mill	48	81	18	140	Normal	100	350	6	2	-	Good
9	18662	Sumaiya	28	F	6	-	-	Teacher	46	65	15	187	Normal	150	370	7	2	+	Good
10	7575	Padma	65	F	1	B	Dust	House wife	58	80	18	168	Normal	160	350	6	1	-	Good
11	17061	Venkadachalam	77	M	36	S & A	S	Agri labour	52	72	17	126	Bronchitis	90	200	5	2	-	Fair
12	1028	Madathi	60	F	12	T	H.G	Agri labour	60	87	24	124	Normal	110	280	6	3	-	Fair
13	18952	Mariyaselvam	28	M	1	S	H.G	Mill	45	78	14	127	Normal	180	350	7	2	-	Good
14	74584	Utchimahali	50	M	9	A & B	H.G	Agri labour	60	80	15	192	Normal	110	360	8	3	-	Good
15	1828	Sandanamariyappan	18	M	5	-	Dust	Auto driver	45	86	15	145	Normal	130	320	6	1	+	Good
16	74956	Gopi	17	M	2	-	Dust	Student	40	78	17	121	Normal	150	380	5	1	+	Good
17	75210	Aliyar	60	M	18	S & A	-	Beedi	40	106	25	164	Normal	80	160	9	6	+	Poor
18	6038	Vijaya ragavan	50	M	12	S	-	Teacher	50	81	26	128	Normal	80	240	5	2	-	Fair
19	76415	Aathi narayanan	44	M	8	S & A	H.G	Agri labour	56	92	21	195	Normal	140	320	6	2	-	Good
20	6615	Jeya	37	F	4	-	-	House wife	45	82	13	189	Normal	120	350	3	1	-	Good

H.G - Husks Of Grains
D & C - Dust & cold Exposure
H.G & CE - Husks of grains & cold exposure
S - Smoker

T - Tobacco Chewer
B - Betelnut chewer
A - Alcoholic
A & S - Alcoholic & Smoker

(-) Negative
(+) Positive

Table - 27 -D

Table - 27 -D																									
S. No	OP. No	BLOOD INVESTIGATION												URINE ANALAYSIS						MOTION					
		BT				HB %	AT				HB %	BT		AT		BT			AT			BT		AT	
		TC	DC				TC	DC				ESR MM Hrs		ESR MM Hrs		Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst
			P	L	E			P	L	E		1/2	1	1/2	1										
1	6950	8500	60	28	12	70	9000	65	31	4	79	7	14	2	4	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
2	1505	9200	55	40	5	76	9800	58	41	1	81	4	8	1	2	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
3	74450	8200	62	34	4	74	9200	62	34	2	78	4	7	2	4	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
4	73049	8500	55	40	5	72	9700	57	41	2	76	3	7	2	4	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
5	9379	8000	52	40	8	70	9200	56	41	3	81	25	55	8	14	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
6	72140	9000	58	28	14	75	9800	60	30	10	85	25	50	20	40	Nil	Nil	2-3 pus cells	NIL	NIL	NAD	NIL	NIL	NIL	NIL
7	698	8300	50	38	12	71	9500	54	42	4	76	14	18	4	6	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
8	18925	8900	64	33	3	72	9800	65	34	1	80	7	14	2	4	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
9	18662	8200	48	48	4	76	9500	56	43	1	81	5	10	2	4	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
10	7575	8000	68	28	4	71	9000	70	29	1	80	5	10	1	3	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
11	17061	9000	62	30	8	73	9800	64	32	4	79	10	18	5	10	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
12	1028	9800	63	25	12	68	10300	66	29	5	80	12	26	6	14	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
13	18952	7900	58	38	4	60	9000	58	40	2	75	15	30	4	8	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
14	74584	8000	68	27	5	73	9800	69	30	1	79	15	28	5	8	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
15	1828	9600	65	30	5	79	10500	66	32	2	83	12	14	3	7	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
16	74956	9000	58	39	3	74	9800	59	40	1	87	2	7	1	3	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
17	75210	9200	50	36	14	75	9600	53	39	8	78	26	52	16	25	Nil	Nil	Few pus cells	NIL	NIL	NAD	NIL	NIL	NIL	NIL
18	6038	9400	60	34	6	81	9800	60	37	3	85	12	15	6	10	Nil	Nil	1-2 epi cells	NIL	NIL	NAD	NIL	NIL	NIL	NIL
19	76415	8600	58	38	14	82	9800	65	41	4	83	20	35	4	8	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL
20	6615	9000	60	34	6	76	10200	61	37	2	80	6	13	2	5	Nil	Nil	NAD	NIL	NIL	NAD	NIL	NIL	NIL	NIL